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EDITORIAL

THE National Society for the Prevention of Tuberculosis have shown great wisdom in publishing a verbatim report* of the Discussion held under its auspices in July 1945. The meeting was held to consider the problem of "The Aftercare and Reablement for the Tuberculous," and was addressed by speakers from industry, specialists in tuberculosis, representatives of local authorities and the Ministry of Labour, and others with special interests in the subject.

The failure to deal with the employment of the tuberculous person after his discharge from institutional treatment has been recognised for the past twenty-five years as the fundamental weakness in our tuberculosis schemes. It is no exaggeration to say that, while the provision and standard of residential treatment has improved steadily though unevenly, and that diagnosis has been revolutionised, only the surface of this problem has been scratched; even this has been achieved mainly by individual endeavour.

The main difficulty was that there was no official recognition that the patient discharged from a sanatorium was usually unfit to compete successfully with a fit individual in the labour market without detriment to his health. Hence, when the patient returned from institutional treatment he and his family usually either had to exist on provision designed for temporary unemployment or sickness, or if he was fortunate enough to obtain employment its circumstances were seldom suitable for his physical condition.

The Medical Research Council Report published in 1942 put the urgency of this problem fairly and squarely before the Government and the public.

The first result, and one of which the value, as far as it went, was recognised by medical speakers, was the introduction of the Tuberculosis Treatment Allowances governed by the Ministry of Health Memorandum 266/T. Its use, however, is limited to giving the patient a breathing space in which to try and regain his foothold in society. The next step has been the passing of the Disabled Persons (Employment) Act. It is on the provision of that Act that the Government propose to rely for the solution of the problem. At the conference a valuable résumé of the powers of the Government under the Act was given by Mr. Gomme, an Assistant Secretary of the Ministry of Labour and National Service. As he indicated, there are three main provisions: industrial rehabilitation, assistance towards employment under ordinary working conditions, and provision of facilities for employment under special

* "Pathways in Aftercare," N.A.P.T., 1945. Price 5s.

sheltered conditions. The first is not likely to be of great value, as the tuberculous persons who are eligible for its benefit are those who, when there is little unemployment, are most likely to find suitable work without assistance; the second, which includes the quota of disabled persons which it will be obligatory for an employer to employ, suffers from the same disadvantage; it is certain to be more convenient for him to employ men disabled for other reasons than tuberculosis. These points were freely acknowledged by Mr. Gomme. It is really on the third that the Government are depending, though its practical application has not started properly, and indeed the sketchiest outline has not yet been drawn.

The proposals appear to include the provision of similar schemes to Papworth, and the setting up of workshops on the lines of the Spiro workshop with ancillary facilities assisted by, or possibly managed by, a corporation set up for the purpose. Mr. Gomme ended up by an appeal for assistance to the officials of the Ministry of Labour in placing tuberculous persons in suitable employment. It was ominous, however, that the most concrete proposal was the issue of the form to be filled up by doctors.

Mr. Gomme's exposition of the Ministry of Labour's plans was followed by a really constructive speech by Mr. Peter Fraser, Managing Director of Papworth and Enham Industries; a breath of fresh air must have been felt throughout the assembly, for here was the practical enthusiast. No one who is interested in the subject should fail to read the speech. It is followed by reports of shorter speeches by industrialists and their efforts to fit tuberculous persons into their factories. A great deal of thought and trouble has been taken by a number of large firms in fitting in ex-patients to suitable work, and as the schemes develop and become more numerous many tuberculous individuals will benefit.

The practical difficulties were put forcibly by a number of Tuberculosis Officers, who laid particular emphasis on the problem of the "good chronic," the high resistance type who can hold a fairly easy job without much deterioration but who is always coughing up tubercle bacilli. It is this type for whom workshops and colonies are most useful, but who, as they usually feel well, generally want to return to more normal conditions.

A shadow, however, hangs over the whole proceedings. The speech of the representative of the Ministry of Labour appeared to lack enthusiasm. He said in effect that he was there to explain what the Ministry could do and report practical suggestions. There was no indication that training centres would be set up to place the tuberculous individual in specially reserved jobs of all suitable types in the Civil Service.

The Civil Service and Local Authority Service are a growing industry and include thousands of suitable jobs. Its offices are spread all over the country; they are convenient for transport; in peacetime the hours are regular, the tenure secure, and usually the work does not involve heavy physical exertion; full wages could be paid for whatever hours are recommended medically. In fact, here is the ideal type of employment in which to employ ex-patients.

Actually here is generally the most difficult employer from the point of view of the Tuberculosis Officer. Even when it is desired to introduce at the bottom an ex-patient who can fulfil all examination requirements and who can

be certified as fit for full-time work, he is usually debarred by being a poor risk for pensions and superannuation schemes. Apart from anything else, the introduction of a hundred tuberculous ex-patients into the Ministry of Labour offices throughout the country would do much to improve the keenness of the Ministry officials to place the sufferers in employment.

It seems an unkind thought, but it is difficult to avoid the feeling that the occurrence of tuberculosis in a number of the working members of the families of high officials in the Ministry of Labour would produce a personal interest in the rapid development of official ideas into practice.

The pamphlet deserves the careful study of those who have to deal with tuberculous patients, as it gives a broad view of the proposals put forward towards the solution of a difficult problem.

F. H. Y.

GENERAL ARTICLES

SURGICAL TECHNIQUE IN THORACOPLASTY WITH PARTICULAR REFERENCE TO SEPSIS

By F. RONALD EDWARDS

From the Chest Surgical Centre, Liverpool

The first stage of a modern thoracoplasty with apicolysis entails one of the greatest tissue exposures of all major operations. Interventions within serous cavities have a certain safety factor superimposed upon them by the fact that the limiting endothelium effuses a large volume of fluid, rich in antibodies and macrophages, which can overcome minor degrees of soiling and infection. This is not so with operations undertaken amongst the tissue planes, for here the exudation is relatively less in amount, and organisms can lurk and multiply amongst the irregular surfaces of the boundaries of the wound without coming into effective contact with the tissue fluid. Such an infection in a first stage thoracoplasty will severely jeopardise the success of the operation, and in a considerable number of cases will prevent its ultimate completion. The extrafascial space normally depends for its obliteration upon the development of an effusion, which is produced as a secondary response to trauma and the presence of blood. The fluid part of this effusion is gradually reabsorbed, leaving behind a fibrin matrix, which undergoes a final organisation, firmly fixing the muscles of the chest wall and the scapula to the rib periosteum and endothoracic fascia. When infection occurs, the amount of effusion is excessive, lifting the scapula and muscles far away from the lung, and in such cases, unless energetic aspirations are performed and some bactericidal agent is inserted, actual rupture through the wound takes place. This leaves a cavity empty of all material except air, and surrounded by bony structures, some of which cannot be dispensed with, and provides a most serious problem in the undertaking of its closure. Furthermore, it is a frequent occurrence for the coccal infection to be followed at a later date by an extension of the tuberculous process into the extrafascial space, either directly from the lung or from the tuberculous intercostal or paravertebral glands which are constantly noted in performing the operation. When a tuberculous infection occurs in such a cavity its final obliteration is almost a hopeless proposition. It is probable that minor degrees of tuberculous infection of the extrafascial space do occur from the above causes in many thoracoplastic operations, but this process is a slow one, and if the fibrin matrix has developed and organisation occurred, then it is localised by the fibrous tissue, and no adverse effect is noted.

It has been my experience that most cases of wound or space infections start with an organism of low virulence. It is usually only later when drainage has been instituted as a result of failure of treatment by aspiration or when a sinus has developed that the more virulent and less easily destroyed organisms

invade the area. If a virulent infection occurs at the onset, this is probably due to a major breakdown in aseptic technique. The more common cause of the primary infection would, however, appear to be due to some minor and possibly unrecognised error in which skin organisms are introduced, as by inadequate sterilisation or protection of the patient's skin or from a tear or puncture in the gloves of the surgical team, including the theatre sister.

After reviewing the course taken by these cases of infected wounds and spaces it was considered that the usual surgical technique, seemingly satisfactory for intrapleural procedures, could not be completely relied upon for thoracoplastic operations, and that a reconsideration of the whole subject in the light of the observations made above would have to be made.

The problem was approached from three angles:

(*a*) The preparation of the patient so that the highest degree of resistance to coccal infections should be developed prior to the operation.

(*b*) The sterilisation and protection of the skin.

(*c*) The elimination of contact of the gloves of the whole operating team either directly or indirectly with any part of the operation wound.

PREPARATION OF THE PATIENT.—The resistance of a subject to infection by organisms and the ability to localise an infection, once established, depends upon the maintenance of certain factors not yet completely elucidated, which collectively might be termed " good general condition." The ability to heal wounds rapidly and firmly by active fibroplastic proliferation and collagen formation is intimately connected with this state. In pulmonary tuberculosis, which is characteristically described as a wasting disease, the changes in the blood and tissue fluids of a chronic toxæmia are frequently manifest, and in particular is this seen in the " slipping chronic " type of case whch forms a fair proportion of those on which a thoracoplasty is called for, after various other collapse measures have been given an unsuccessful trial. Clinical anæmia, hypoproteinæmia, and avitaminosis may actually be present, but often this is not so and the real deficiency is in the body reserves of iron, protein and vitamins, upon which such a call is made in the immediate after-stages of an operation. In the case of a thoracoplasty where two or three extensive procedures will follow at fortnightly intervals, the drain is tremendous. The administration and utilisation by the body of these materials takes time, and the giving of them in between each operation does not appear to produce the same effect as preoperative saturation. Special emphasis is laid upon the blood proteins, and the level should be as high as possible in view of the fact that the gamma globulins are now shown to carry the antibodies of the blood stream,[1] in addition to the essential osmotic value of the albumin factor.

The erythrocyte and hæmoglobin level is raised to as near normal as possible by the administration of hæmatinics, of which I find mist. ferri and ammon. cit. the most rapidly absorbed, and if necessary blood transfusions. Not only is the high hæmoglobin level necessary for sound wound repair, but also for maintaining circumstances as favourable as possible for the action of a myocardium, which in many cases has severely suffered under a prolonged toxæmia. Vitamin saturation, especially with C, is advisable, as deficiency in this factor leads to poor collagen formation.

Adequate stores of carbohydrate and protein in the liver and tissues is ensured as far as possible by the administration of a high calorie-high protein diet for four weeks before the operation. The diet given in the Appendix is adapted to the present system of rationing; 3,000 calories and 100 grammes of protein per day are taken. Extra glucose drinks for one week preoperatively provide carbohydrate abundance.

With the more advanced cases normality of the blood and tissue chemistry may be impossible of attainment, but the efforts made will render more likely the final success of the operation.

Sterilisation and Protection of the Skin.—For ten days prior to the operation the skin of the operation area is painted with Viacutan (Ward Blenkinsop and Co.) every other day and a sterile jacket worn. Viacutan is a silver preparation with a satisfactory skin-sterilisation effect, and has the advantage that it can be used over a long period of time without producing skin irritation, although a slight degree of exfoliation of the horny layer may occur. It is of great value in those patients who have a tendency to a folliculitis, and is the best material we have yet found in rendering these skins surgically approachable. The jacket is made of calico with an arm-hole on the operation side, and fastened with tapes in the opposite axilla. A normal skin preparation is made on the morning of operation, with a final application of iodine. Iodine is used for preference as an immediate preoperative application, although if there is skin sensitivity to this Viacutan is used throughout. The usual technique of having all towels and tetra cloths lined with an impermeable material such as rubber or batiste is employed, so that no skin secretions can soak through, particularly if they become wet.

"No-touch" Operative Technique.—The actual surgical technique is similar to that described by Watson-Jones[2] for operations in orthopædic conditions, in which the same problem of sepsis with resultant crippling deformity had arisen. It is assumed that the gloves of the operating team, including the theatre sister, may not be sterile due to minute punctures. The instrument table is divided horizontally into two halves with a coloured towel, and the coloured area is inviolate. The handles of the instruments go on to the white part of the table nearest to the operator, and the parts that come into contact with the wound on to the coloured portion. The sister handles all instruments after sterilisation with Cheatle's forceps, and on no occasion does she touch the handles with these. The instruments are not cleaned during the operation, as this procedure may spread organisms to the working end from the handle. All sutures are threaded and tied on to the needle with forceps, suturing performed with needle holders, and knots tied with artery forceps. Diathermy is used as much as possible to control bleeding, but where ligatures are necessary these are applied and tied with forceps. The technique is facilitated by the use of long instruments throughout. Certain manœuvres during the operation should be stressed. The knife that makes the skin incision or excises the scar of the previous operation when the second stage is being done is abandoned once the deep fascia is reached. The artery forceps that clamp the superficial vessels are all resterilised after being used once, as they tend to come into contact with the skin surfaces before the tetra cloths are applied. All swabs are applied with forceps or holders, and do not touch the

gloves. When the skin is being sutured a separate needle is used for each suture and then abandoned.

No bactericidal agent is put into the wound as a routine, but occasionally, where it has been impossible to get the skin of the back into a reasonably good condition, a little sulphathiazol powder has been sprinkled into the wound.

To make up for protein loss during the operation, a plasma drip is given into the arm in association with the intravenous barbiturate drip, which is routinely given as an adjunct to the local anæsthesia. If blood loss is considerable, then blood is given as well.

In description the technique may appear to be somewhat complicated, but once it has been mastered its employment becomes delightfully easy, and no time is added to the operation.

Results

The full technique has been in use for the last thirteen months on all cases in two sanatoria, during which time 126 thoracoplastic operations were performed on 55 patients. One case of superficial wound sepsis occurred in this series, which delayed the interval between the first and second stage by fourteen days. This was due to a *Staphylococcus albus* infection without pyrexial response, and responded immediately to penicillin applied locally.

The details of the cases are as follows:

Cheshire Joint Sanatorium.—89 operations on 40 patients (38 suffering from parenchymatous disease and 2 from tuberculous empyemata):

1st Stage—38 operations (24 extrafascial apicolysis).
2nd Stage—36 operations.
3rd Stage—9 operations.
Ant. Stage—2 operations.
Revisions—4 operations (1 death from pneumonia).

Two of these patients developed small wound breakdown, one fourteen days and one two months after completion of the thoracoplasty. Both disruptions were sterile and probably due to pressure of the splint. Healing occurred in a few days.

Wrightington Hospital (*Lancs. C.C.*).—36 operations on 15 patients (all parenchymatous disease):

1st Stage—15 operations (6 extrafascial apicolysis).
2nd Stage—14 operations.
3rd Stage—5 operations.
Ant. Stage—1 operation.
Revision—1 operation.

Two deaths occurred in this series, one after the first stage and one after the second stage, due to bronchopneumonia in high risk cases. Death occurred in each case after seven days, and there was no evidence of wound infection during this time.

Discussion

The incidence of sepsis and its probable grave consequences in thoracoplasty wounds is well recognised by thoracic surgeons, as is shown by description of methods of dealing with the problem published in the literature. Deryl Hart[3] in 1936 described the improvement in his results as a result of operating under ultraviolet light rays, and in 1940 Overholt and Betts[4] supported the value of this procedure. These latter authors showed that their original infection rate, which was 13·8 per cent., was reduced to 6·53 per cent. by the use of individual skin needles, and later to 2·67 per cent. by the use of ultraviolet light. In 1943 Cory[5] rubbed sulphathiazol powder into all exposed raw areas, and in 90 thoracoplastic operations had 5 incidences of " slight stickiness of the stitch holes " and 1 incidence of " deeper sepsis than superficial remaining localised." Wu and Pianetto[6] in 1943 described 120 thoracoplastic operations in 55 patients without any sepsis. Air contamination was dealt with by keeping as much of the wound covered as possible with towels, and at the end of the operation irrigating the entire wound surface with warm normal saline. Great care was taken with the preoperative " build-up " of the patient, and their final conclusion was that, by careful application of the general principles of surgery, the incidence of sepsis can be reduced to a minimum.

No attempt was made to deal with air contamination in the 126 operations reported above, except to use a pack to cover the parts of the wound nearest to the operator, with the more essential idea, to my mind, of preventing the gloves coming into contact with the tissues. An impermeable cellophane mask is worn by all members of the operating team to prevent the possibility of nose and throat organisms entering the wound. No air conditioning or air sterilisation is used in the theatres in which these cases have been operated upon.

In conclusion, I must agree with the observations of Wu and Pianetto that the meticulous application of general surgical principles both before and during the operation of thoracoplasty is the fundamental principle in the elimination of sepsis. This has resulted in the reduction of the rate of sepsis to below 1 per cent., a figure which in my hands is a great improvement on previous results, and on my figures at another centre where I performed thoracoplasties during the same period, but did not utilise the particular operative technique described.

Summary

Sepsis in thoracoplasty is a serious hazard and may prevent the completion of the operation.

The fundamental factor in its elimination is the meticulous application of general surgical principles. The resistance of the body to infection is built up before operation. A prolonged skin preparation is performed. The operation itself is performed completely under a " no-touch " technique.

The application of these principles has lowered the rate of sepsis to below 1 per cent.

My thanks are due to Dr. Peter Edwards, Medical Superintendent of the Cheshire Joint Sanatorium, and to Dr. J. Dobson, Medical Superintendent of

Wrightington Hospital (Lancashire County Council), for permission to publish these results. I am grateful also to Miss Jean Keay, Dietitian at the Clatterbridge (County) General Hospital, Bebington, Cheshire, for preparing the high protein diet for these patients.

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Appendix

DIET FOR THORACOPLASTY CASES

3,000 *Calories* Each Day.	100 *gms. Protein* 2 *pints milk* for special cases (T.B. and post-major operations). (1¾ pints per patient to be sent to ward, ¼ pint to be used in the kitchen for puddings, sauces or soups). 1 *oz. fat* for use in ward (6 ozs. butter and margarine and 1 oz. dripping per patient per week).
Before Breakfast.	If desired, a cup of tea with milk.
Breakfast.	Tea with milk, sugar if desired. A large serving of porridge and milk (about ¼ pint). A varied breakfast dish (see below). Bread or toast (about 3 ozs.). Margarine. Marmalade at least once per week. Syrup, etc., as available.
Mid-morning.	A milk drink (½ pint milk).
Mid-day.	1-2 oz. meat, etc. (see below). 8 ozs. potatoes (raw weight). Vegetables (see below). Steamed pudding and sauce, or milk pudding, or stewed fruit with custard, or milk pudding or pastry, baked pudding, etc. (¼ pint milk).
Tea.	Tea with milk and sugar. Bread and butter or margarine. 1 or 2 of the following: Jam, cake, lettuce, watercress, tomatoes, biscuits.
Supper.	½ pint milk as milk drink (or to be used in a pudding). Savoury on pudding, etc. (below). 2 ozs. bread or toast. Margarine or dripping.
Bed-time.	½ pint milk.

Breakfast.	Suggested variations: 1 egg or dried egg dish once per week. Fish cake and baked or fried potatoes twice per week. Bacon and baked or fried potatoes twice per week (or bacon once and a boiled bacon dinner). Sausage, herring, kipper, haddock, or dried egg dish twice or three times a week.
Mid-day.	Suggested variations: *Meat* can be served as stews, roast, or rabbit, ham, hot pot, sausage meat and bacon rolls, meat and potato pie, etc., on five days per week (when roast meat is served it is better to allow 2 *ozs.* cooked meat per person and to give a *vegetable* stew on one of the other days instead of trying to make the roast do two dinners). Sausage or sausage meat once per week. Fish (about 6 ozs. cooked) once per week.

Vegetables:
Greens—three times per week.
Carrots or swedes—at least twice per week (once as separate vegetables).
Root vegetables, peas, beans, lentils, etc., in stews, etc.—twice per week.

Supper. Suggested variations:
1 oz. cheese and a vegetable soup.
A cheese dish, pilchards, sardines, baked beans, etc.
Sweet or savoury custard (dried eggs).
Made-up dishes, including potatoes and dripping (*e.g.*, roast potatoes stuffed with sausage meat).
A milk pudding and stewed fruit.
Thick vegetable soup and raw apple or salad, etc.

RATIONS FOR SEVEN DAYS

	Amounts.
Milk	14 pints
Meat (cooked)	about 6 oz.
Eggs (at least 1 dried)	2
Fish (½ lb. cod, etc., ½ lb. herrings, etc.)	1 lb.
Sausage	2
Cheese (according to ration)	2 oz.
Margarine and cooking fat (ration)	6 oz.
Butter (ration)	2 oz.
Sugar (ration)	8 oz.
Bread (at least 3½ lbs.)	3½ lb.
Potatoes (uncooked)	7 lb.
Bacon (ration)	4 oz.
Tinned fish	2 oz.
Jam (ration)	4 oz.
Cabbage or other fresh greens	1½ lb.
Oatmeal (for porridge)	8 oz.
Flour, cereal, etc.	10 oz.
Rice	2 oz.
Barley	1 oz.
Cocoa	2 oz.
Dried peas, etc.	at least 4 oz.
Dried fruits	at least 4 oz.
Carrots, swedes, etc.	at least 1 lb.
Cake	6 oz.
Salad, tomatoes, etc.	as much as possible

The amounts give 106 gms. protein (about 50 gms. animal and about 50 gms. vegetable), and 2,920 calories.

PULMONARY TUBERCULOSIS AND PREGNANCY

BY RAYMOND C. COHEN
From Black Notley Hospital

ONE of the most controversial subjects in medicine is the relationship between pulmonary tuberculosis and pregnancy. When it is remembered that in this combination of pathological and physiological processes we are dealing with factors each of which is highly variable, it is not surprising that their effects should be difficult to assess. So the pendulum of opinion has swung from one extreme to the other. It is said that Hippocrates and Galen actually advocated pregnancy as a treatment for pulmonary tuberculosis; and until the middle

of the nineteenth century it was generally believed that a phthisical woman's health improved during gestation. About this time, however, a complete reversal of opinion occurred which sometimes went so far as to say that every pregnancy in a tuberculous woman, even though her disease be quiescent, should be terminated by therapeutic abortion. More recently the pendulum has seemed to take a neutral position. Certainly the advances in diagnosis and treatment of the last twenty-five years have made necessary a review of the position, for a clear answer to the problem of the effect of pregnancy and all that it entails for the phthisical woman has yet to be made.

Review of the Literature

When one considers the pessimistic attitude of the medical profession as a whole to the subject of tuberculosis and pregnancy, it is a revelation to find that the majority of papers on the subject have a much more hopeful outlook.

Oxenham (1941), in a recent comprehensive and critical review of the literature, records his astonishment on the unanimity of opinion expressed by tuberculosis workers. In his own words: " They unanimously express the opinion that the tuberculosis should be the first object of the treatment, and that therapeutic abortion should rarely, if ever, be employed, because of its great inherent danger, and because results equally good can be obtained without sacrificing the life of a healthy baby for a dubious benefit to an unhealthy, and perhaps in any case doomed, mother."

Brooks (1940) states that " a close study of the literature yields some amazing facts, the most outstanding of which is that scarcely any scientific data exist by which the hypothetic effect of pregnancy on pulmonary tuberculosis may be evaluated."

Krause (1935) makes some pertinent remarks on this theme. He particularly refers to the unjustified assumption that all women have an equal tolerance to pregnancy. He concludes that the good or bad influence of pregnancy on tuberculous women is determined by whether they would have tolerated pregnancy well or poorly if they had not been tuberculous.

Brooks considers that arrested cases of tuberculosis may proceed to term " with reasonable safety," that active cases should not " be aborted on principle," and that abortion after the fourth month involves as great a hazard as labour at term. He states: " There is reason to suppose, therefore, that with expert management the prognosis for patients in whom pulmonary tuberculosis and pregnancy coincide is by no means gloomy." In spite of this, and contrary to his previous arguments, he advocates abortion for " many cases of active phthisis which are either pregnant for the first time, or which give the history of tolerating pregnancy poorly." He does not justify this latter conclusion, which lacks the authority of supporting evidence.

Some valuable experimental work has been done on this subject. Thus Burke (1940) carried out experiments on rabbits; 134 were injected intratracheally with human tubercle bacilli, and 106 with bovine tubercle bacilli. Nine of the first group had litters amd twelve of the latter (most of them had repeated pregnancies). Only one actually died of tuberculosis, and the others were killed " to permit of study of the lesions at different stages of the disease." Comparison of X-ray and post-mortem findings in the pregnant rabbits with the findings in males and non-pregnant females did not reveal anything to suggest that pregnancy had had an influence on the progress of the tuberculous infection.

Wade, also (1942), using rabbits infected with bovine tubercle bacilli, found that the expectation of life in a pregnant group of 23 rabbits was somewhat longer than in a control group of 20 non-pregnant rabbits. The average extent of disease at death was less in the pregnant than the control animals. When his figures were subjected to statistical analysis the difference was found to be too small to be significant, but at least no harmful effect of pregnancy was demonstrated.

With regard to the endocrine disturbances in pregnancy, Steinbach and Klein (1937) carried out some interesting experiments on rabbits and guinea-pigs artificially infected with tuberculosis, and concluded that " experimental tuberculosis in rabbits and guinea-pigs was favourably influenced by the administration of antuitrin ' S,' pregnant mare serum, and to a less extent follutein. No retardation of disease was obtained by the use of either anterior pituitary extract or emmenin. The results suggest that the gonadotropic hormone may be a factor in the temporary amelioration of symptoms observed in tuberculous women during pregnancy."

A number of writers have attempted to assess the influence of pregnancy on pulmonary tuberculosis from the study of statistics. Thus Edson (1942) points out (New Zealand figures) that whereas the death-rate from respiratory tuberculosis tends to be higher for single than married women (this may be due to the generally improved living conditions and financial status of the latter), " the mortality of married women is fairly uniform through the main child-bearing years, but reaches a maximum at twenty-five to twenty-nine, suggesting that latent tuberculosis flares up under the influence of the first pregnancy." Unless it is accepted that the majority of first pregnancies occur in this age group, it is difficult to see how this deduction can be justified.

Again, the striking fact that young adult females did not share in the reduction of tuberculosis mortality which occurred since the beginning of this century is connected by Wolff (1939), on statistical evidence only, with maternal activity. By correlating birth-rates and mortality from tuberculosis, he hypothesises that the risk of young females had not diminished in the same degree as that of older women by virtue of the strain placed on them by child-bearing. This argument, though apparently statistically justified, does not take into account the many other factors which influence the progress of the tuberculous patient. Moreover, in this country the slowing-down in the decline of tuberculosis also occurred among young males, though admittedly to a much lesser extent than among young females. Endocrinal changes associated with adolescence may be important.

Hart and Wright (1939), however, showed statistically that the young women age group has been sensitive to changes in the national standard of living, and that a retardation in the improvement of this standard since the beginning of the century has been a major factor in slowing up the mortality decrease for this group. This retardation was accompanied by a falling-off of the improvement of housing conditions of this country as a whole, which continued till after the 1914-18 war. The increasing proportion of young women entering gainful employment was also shown to be a factor, and during the years 1914-18 the lowered standard of living, with food shortage, overcrowding, and increased industrialisation, was sufficient to accelerate the retardation in decline of phthisis mortality in young women into an actual severe, though temporary, rise. The evidence of Wolff's theory of maternal strain is puny and unimpressive compared to the solid statistics and reasoned deductions of Hart and Wright in their comprehensive monograph.

In a careful analysis of 451 married women admitted to the Trudeau Sanatorium, Jameson (1938) found no significant difference in the death-rate from tuberculosis in patients (approximately 12 per cent.) whose tuberculous disease was related to a full-term pregnancy, as compared with that of a group of nulliparas of the same age group and showing lesions of similar extent. This is an important observation, though as Jameson himself points out, his patients were culled from a social class which returned to favourable conditions on discharge from sanatorium, and were unlikely to be subjected to the strain of domestic work and household cares. His findings, however, hold good in so far as the effect of pregnancy and labour is concerned. Social conditions are accepted as of first importance to the progress of a tuberculous patient, but it is right to make a clear distinction between the effect of these and the effect of pregnancy. Whether termination of pregnancy is justifiable because it is known that, although the woman could be expected to go through pregnancy and labour without harm, she would subsequently be returning to unfavourable living conditions is a highly debatable point that can only be decided on consideration of individual cases.

Alice Hill (1928) carried out a careful and exhaustive review of 349 pregnant tuberculous women, and compared these with a control group of 160 tuberculous women who had never been pregnant or whose pregnancies were in no sense associated with their tuberculous disease. The pregnant cases were reviewed one year after delivery. She concluded that pregnancy had no appreciable bearing upon the progress of the tuberculous disease, that the danger of primiparity among the tuberculous is similar to that among mothers in general, and that the maternal mortality rates among the 349 cases corresponded with the death-rates of non-pregnant tuberculous women. Hill also pointed out that only one-third of the pregnant women had had as much as six weeks' sanatorium treatment, and it is important to note that, according to her figures, 148 of her cases were not diagnosed till after pregnancy. One in three of these cases was dead one year after delivery.

Ornstein and Epstein (1939) point out that a critical analysis of statistics does not support the belief that pregnancy activates the tuberculous process, and that much of the current divergence of opinion is due to the high death-rate from pulmonary tuberculosis among females in the child-bearing age. They cite a death-rate of 33 per cent. among non-pregnant females (1,805 deaths in 5,469 patients), which they compare with a death-rate of 36 per cent. among 85 pregnant tuberculous women. While the comparison of these two groups may be open to criticism on statistical grounds, the figures are interesting, and the authors report a further 82 cases of pregnancy and pulmonary tuberculosis. Of these, 62 cases were classified as " improved " on discharge, and pneumothorax therapy was used in 33 cases. Ten deaths occurred, all in patients with caseous pneumonic disease. They concluded that " pregnancy has no influence on the course of pulmonary tuberculosis."

Lyman (1943) makes a statistical analysis of the follow-up results of 1,818 tuberculous women, dividing them into three groups—782 married before admission to sanatorium, 315 married after discharge, and 721 remaining single. The follow-up period was variable, but 1,092 were reviewed after four years and 206 after twenty years. Lyman makes the striking point that the group marrying after treatment (he calls them " later married ") had a prognosis four times better than the single group, and three times better than those married before treatment. (That marriage tends to improve the prognosis of quiescent or stabilised cases has been referred to by several writers,

and is probably due to improved social and domestic factors.) 192 of the 315 "later marrieds" had histories of pregnancies, averaging 2·25 each, and Lyman suggests that the better record of this group is due to their not having undertaken pregnancy until after their tuberculosis had been brought under control, and they had themselves been trained to take proper care of their health. He also states that when reactivation of the disease occurred "in close relation" to pregnancy, "it did so without exception either during pregnancy or in the three months after delivery." Where active disease preceded the pregnancy the mortality was 14 per cent., where it occurred during pregnancy the mortality was 78 per cent., and when it occurred during the year following delivery it was 60 per cent. He concludes that "especially in younger women, pregnancy contributes a grave risk to an active case of tuberculosis," but goes on to say that since gathering these statistics he has been more and more impressed by the good results of collapse therapy, and advocates hospitalisation with such treatment as an alternative to abortion.

Among those authors who believe pregnancy to have a harmful effect, Rist (1927) reports the death of 86·5 per cent. of 52 pregnant tuberculous women within two years of their labour, but his paper loses authority from the lack of information of the types of disease and of treatment provided.

Sergent (1926) obtained a history of pregnancy eight to ten months before onset of symptoms in about 25 per cent. of a group of tuberculous women.

Bernard (1921), from a review of 327 patients, satisfied himself that pulmonary tuberculosis frequently arises or becomes aggravated during or soon after pregnancy.

Norris and Murphy (1922) studied the results in 166 cases. They concluded that pregnancy has a deleterious effect on tuberculosis, but their figures are not convincing, as the difference in results between the non-pregnant and pregnant group is not significant. Actually, when followed up, the mortality of the non-pregnant group of their "Stage III" cases was about twice that among those who had had pregnancies, though again the figures are too small to be significant.

Pollak and Potter (1940), in a brief review of 97 cases, of whom 52 suffered from active pulmonary disease, concluded that pregnancy is harmful unless adequate rest and care are provided, and that the late results in quiescent cases are worse than in a comparable non-pregnant group. The paper lacks detailed information, particularly of treatment provided and of follow-up records.

In one of the few papers in the literature giving clinical details, Cutler (1944) considers that pregnancy has an unfavourable influence. Reporting on 26 cases of active disease, he stresses the efficient protection afforded by adequate collapse therapy, so that "women in whom this was true came through pregnancy without harm and were able to lead normal lives the same as any non-tuberculous mother." (Whether a patient still receiving collapse therapy should be allowed to lead the same full life as her non-tuberculous sister is debatable.) Cutler concludes that patients with advanced disease, or with active disease which does not respond to collapse therapy, do badly.

Phyllis Dingle (1944), accepting that pregnancy has a harmful effect on phthisis, claims to have prevented a breakdown in the puerperium in 27 out of 30 patients by inducing a pneumoperitoneum, to prevent a "fall of the diaphragm," within an hour of delivery. 3,000 c.c. of air are introduced at the induction. The article is not convincing, as she produces no real evidence that the puerperium would otherwise have been attended by breakdown,

neither does she show, by comparative measurement, that the pneumoperitoneum has elevated the diaphragm, or that without it there would have been a significant fall. Most of the 3,000 c.c. of air is surely directed towards distending a naturally lax abdominal wall, and, in fact, the change in level of the diaphragm before and after labour is seldom marked.

Michelis (1937), among others, also advocated the induction of pneumoperitoneum in the puerperium for the same reason. He describes only one case, and the radiographs produced show a good air bubble beneath the diaphragm, but the only structures affected are the abdominal contents, which are displaced downwards, leaving the diaphragm unaffected.

Rist and Jottras (1935) described 132 pregnant tuberculous women treated by artificial pneumothorax. They believe pregnancy and labour to have a harmful effect, but consider this harm may be considerably reduced by pneumothorax therapy. They found that cases in whom the pneumothorax was induced before the onset of pregnancy had a materially better prognosis than those in whom it was induced after.

Snider (1940) believes that pregnancy is harmful to the tuberculous woman, and that its prevention is always advisable. When it has already occurred, however, abortion must not be undertaken lightly because (i) tuberculosis and pregnancy is not always a fatal complication and the fœtus' life may be sacrificed unnecessarily; (ii) abortion may fail to save the mother's life; and (iii) the disease may be so far advanced that termination is useless and there may be a good chance of obtaining a live healthy child.

Against the unfavourable verdict of these writers must be placed the conclusions of others, and to a reader who endeavours to approach the subject with an unbiased mind the evidence opposing the harmful effect of pregnancy seems to be both more extensive and more authoritative. In addition to the statistical and experimental papers already quoted, the following summaries of published works are evidence in support of this.

Marshall (1931) compared 309 non-pregnant and 303 pregnant females in various stages of the disease, and found that of " dormant and healed " cases, 2 per cent. of non-pregnant and 1 per cent. of pregnant died the first year, and of " advanced " cases 46 per cent. of non-pregnant died compared with 37 per cent. of the pregnant.

Seeley, Siddall and Balzer (1940) reviewed 31 thoracoplasty cases who subsequently became pregnant, including 8 of their own. One patient died five days after delivery (active tuberculosis in other lung early in pregnancy), 1 became worse during pregnancy, and 4 became worse post-partum, after intervals between one and two years. They believe that the majority of women may safely go through pregnancy after thoracoplasty, though admit that there is risk in some cases due to reduction of vital capacity and possible cardiac strain.

Jameson (1935), in his book on " Gynæcological and Obstetrical Tuberculosis," makes the severe criticism that he failed to find a single report purporting to show the harmful effect of pregnancy which was not open to criticism on grounds of incomplete data of extent and type of the lesions, treatment provided, ante-natal care, etc. He considers that the alleged harmful effects are more apparent than real, and that the proportion of cases becoming worse after delivery is no greater than would be expected in the natural course of the disease. I agree with Jameson that on the difficult question of " abort or not to abrot " in cases of active tuberculosis, decision should be delayed till the third month. Much valuable information may be gained during this

period of observation and no harm done by the delay. If the lesion is such that the prognosis with adequate treatment would be good if the patient were not pregnant, she should be allowed to continue. With advanced cases other factors arise, but the chances of the pregnancy going to term and a healthy baby obtained are good, and prolongation of the patient's life by abortion cannot by any means be guaranteed. In these cases I believe that the wishes of the patient and her family can with justification be taken into account.

It is in the intermediate cases of active disease, in which complete quiescence of the disease is not to be anticipated, that the decision is most difficult. It is in these cases that an individualistic attitude should be adopted, and each case considered on its merits, taking into account social circumstances, parity, reaction to previous pregnancies, and the intelligence and co-operability of the patient. The very real risk of infection of the offspring if a sputum-positive mother does not adopt proper precautions must also be borne in mind.

Mathews (1940), writing from the aspect of the obstetrical expert, stresses the improved treatment and prognosis of active cases of pulmonary tuberculosis, which has rendered inaccurate or obsolete conclusions drawn by perfectly sincere and able writers ten or twenty years ago. The only effect of tuberculosis on pregnancy is a slightly increased frequency of premature labour, and he comments on the bad prognosis that complicating laryngeal tuberculosis implies. This my own experience supports, and I agree with Myerson (quoted by Mathews) and others that pregnancy is contra-indicated in cases of active pulmonary tuberculosis complicated by severe tuberculous laryngitis. Mathews advocates abortion when "the tuberculosis cannot be properly managed," and in advanced or caseous pneumonic disease he considers that pregnancy, labour, and the puerperium "do not present the hazards they formerly did," with modern methods of treating the tuberculosis. He allows breast-feeding in certain cases with negative sputum for the first eight weeks. This seems a dangerous procedure, as sputum testing is by no means a certain proof of non-infectivity, and he makes no mention of the wearing of a mask by the mother.

Forssner (1924) found that the proportion of pregnant women (185 cases) retrogressing or dying was no more than occurred in a group of 359 non-pregnant women. He believes the harmful effect of pregnancy has been exaggerated and opposes therapeutic abortion.

Schultze-Rohnhof (1932), who has written widely and ably on the subject, concludes that pregnancy, labour, and the puerperium have little influence on pulmonary tuberculosis in most instances, and that this influence has been greatly exaggerated.

Divoux (1931), although believing that an apparently benign tuberculous lesion may retrogress as the result of pregnancy, admits that most cases will not be harmed if adequate sanatorium treatment is provided to term.

Gellhorn (1928) also advocates thorough treatment of the pulmonary disease in pregnant tuberculous women. He points out that the results of abortion leave much to be desired and thinks it rarely indicated, and only in patients who will then have a good chance of recovery. Laryngeal tuberculosis he regards as an exception to this, and an absolute indication for abortion. This seems rather stringent, as many mild cases of tuberculous laryngitis with "treatable" lesions in the lung will recover with proper treatment; and as, at the other end of the scale, severe laryngitis usually accompanies advanced pulmonary disease, and the operation of abortion would seem to be useless if the mother is doomed in any case.

Baker and Ward (1942) report 11 cases of pregnancy and pulmonary tuberculosis, none of whom retrogressed after labour. On this limited experience they conclude that if proper treatment is provided, therapeutic abortion is rarely indicated and that, subject to this, the risk of pregnancy in tuberculous women is no greater than in the non-tuberculous. Collapse therapy of some kind during pregnancy they consider of great value, and practice forceps delivery as soon as the cervix is fully dilated, using local infiltration with perineal block for anæsthesia.

Castlen (1936) also makes similar conclusions and recommendations, but bases his opinions on the reports of other observers taken together with his own experience. His paper reports only 4 cases.

Mariette, Larson and Litzenberg (1942) also believed that " when the tuberculosis is properly treated, pregnancy probably does not adversely affect the tuberculous process." Their paper deals with 86 cases, 64 of whom were delivered in sanatori.

Present Investigation

This present paper embodies the result of an experience of 177 consecutive patients with pulmonary tuberculosis who have been confined in the Maternity Unit at Black Notley. This Unit, opened in 1937, has already been described in a paper (1943) giving a brief review of results in the first 100 cases confined at Black Notley; but a short description is desirable here. No selection is made of the cases admitted, and any woman resident in the County of Essex who suffers, or has suffered, from pulmonary tuberculosis is eligible. Quiescent cases are admitted four to six weeks before confinement; active cases as soon as possible after the diagnosis of tuberculosis is made.

Management of Cases

To quote from the earlier paper, " quiescent cases are encouraged to live what one may term an active sanatorium life—*i.e.*, to walk up to two miles daily and perform the little daily tasks which form part of the normal life of such cases in a sanatorium. Progressive cases are managed in accordance with the extent and activity of their pulmonary disease, and on the usual sanatorium lines, including, where necessary, active treatment such as collapse therapy. During labour forceps are not applied as a routine in the second stage, as has been advocated, but only when a definite indication exists. As a general rule, however, no woman was allowed to go more than two hours in the second stage without their use being considered, and if there were signs of maternal fatigue without satisfactory advance of the head they would be applied." Minnitt's gas and air apparatus is used for anæsthesia in normal labours, and gas and oxygen (occasionally chloroform has been used) for any manipulation.

To quote again, " it was found that an artificial pneumothorax did not cause embarrassment to the mother during labour; neither was it ever necessary to give a refill immediately after labour in order to compensate for the alleged fall in intrapleural pressure which has been thought to occur as a result of the descent of the diaphragm."

It is most important that the psychological management of these patients

be not neglected. Worry is a potent factor in helping to determine the breakdown or extension of pulmonary tuberculosis, and the woman should be encouraged to regard her pregnancy and labour as an important incident in her tuberculous career, but not as a potential source of devastating breakdown, or as a means of conveying the disease to the next generation, still unborn. The latter reassurance is justified by the extreme rarity of true congenital tuberculosis. Whitman and Green (1922) could trace only 47 authentic cases in the literature. Tuberculin testing (Mantoux 1 in 10,000 and 1 in 1,000 dilutions) was performed on a consecutive series of 50 of the Black Notley infants within a few days of birth. The results were negative in all cases.

Care of the Infants

Post-natal infection, however, is a real danger, and when new-born infants are nursed in a ward with adult cases of active pulmonary tuberculosis it is essential to enforce strict rules for their protection. The reasons for these rules are explained to every expectant mother on admission, and at Black Notley these precautions include:

1. No mother (or visitor) is allowed to enter the infants' nursery without permission from the nurse in charge on each occasion.

2. Every mother is shown her baby daily. If the mother is actually or potentially infectious she is not allowed to handle it, but it is shown to her by a nurse at the entrance to her cubicle. Nurse and mother are masked.

3. Breast feeding is allowed only in very exceptional cases, and only in those classified as " recovered," or possibly in quiescent cases of simple tuberculous pleurisy. I have not been convinced that any infant has suffered as a result of this prohibition of breast feeding. (Lactation is suppressed, in most cases with ease, by adequate doses of Hexœstrol, B.P.C.)

4. A mother must *never* approach her own or any other infant unless wearing a mask. This applies equally to active or quiescent cases.

5. The mothers are instructed in the feeding and care of their infants before discharge, and emphasis is laid on the importance of wearing masks, even if their pulmonary disease is believed to be quiescent.

Except in " recovered " cases, it is usual to tell the mother that, after her discharge from hospital, the longer the baby can be cared for away from herself, the less will be the strain on her, and the less the risk of infection to her child.

Results

The cases have been divided into three groups, according to their pulmonary condition on admission to hospital for confinement:

1. *Arrested and recovered cases :* those in which there had been no clinical or radiological evidence of active disease for over two years.

2. *Quiescent cases :* those in which the disease, on all available evidence, showed no evidence of activity.

3. *Progressive cases :* those in which there was clinical or radiological evidence of activity.

A strict standard was adopted throughout, and any laboratory or X-ray evidence of retrogression, however slight, was accepted as being due to the

pregnancy and labour. The immediate results (*i.e.*, within three months of labour) are summarised thus:

TABLE I.

Class of Case.	*No. of Cases.*	*No. showing Retrogression.*
Arrested and recovered	69	3
Quiescent	50	5
Progressive	58	15
Total	177	23 (13%)

These figures, on the experience of 177 cases, are comparable to those previously published of the first 100 cases delivered at Black Notley.

More detailed study of the case-records brings out certain points of interest and importance:

1. Of the 3 arrested and recovered cases which retrogressed, 2 improved before discharge; of the 5 quiescent cases 1 improved; and of the 15 progressive cases 2 improved.

2. Of the 15 progressive cases who retrogressed, 10 were suffering from advanced bilateral pulmonary tuberculosis in whom the prognosis would have been regarded as almost hopeless in the absence of pregnancy. (Cases 78, 82, 86, 133, 134, 137, 145, 156, 172, 177.)

3. Of the 23 cases who retrogressed in all groups, only 3 had difficult or complicated labours. On the other hand, 28 patients who had difficult, complicated, or arduous labours did not suffer as a result.

4. Of 58 cases with active disease on admission, 32 showed improvement after labour, and 12 were quiescent by the time they were discharged. (Cases 83, 94, 95, 96, 98, 100, 132, 135, 141, 143, 154, 171.)

5. Premature labours occurred in only 4 women; in one of these the pulmonary disease was "recovered," and in the other 3 "progressive." There were 2 cases of intra-uterine death before term, and 5 still-births, of which 2 occurred in progressive cases. Except for this possible tendency to premature labour in cases with active disease, pulmonary tuberculosis appears to have little effect on pregnancy and labour.

6. The influence of age and parity is difficult to assess, but Table II summarises the available facts, considering the cases who retrogressed:

TABLE II.

	Arrested and Recovered Cases.		*Quiescent Cases.*		*Progressive Cases.*		*All Cases.*
	Under 30.	*Over 30.*	*Under 30.*	*Over 30.*	*Under 30.*	*Over 30.*	
Primiparæ	2	1	4	2	3	6	18
Multiparæ	0	0	0	0	3	2	5

Approximately half the patients retrogressing were under thirty years of age, and three-quarters were primiparæ. Oft-quoted statements that a tuberculous woman is unlikely to pass through more than one pregnancy successfully are hardly borne out by these results. On these admittedly limited figures it seems that tuberculous multiparæ tolerate pregnancy better than primiparæ. The latter have not had repeated pregnancies to threaten their general health, but they are usually younger and in an age group which has a worse prognosis, so far as pulmonary tuberculosis is concerned, than that of the average multipara.

Table III gives an analysis of the duration of quiescence before labour in the Groups 1 and 2 cases:

TABLE III.

Quiescent Interval.	*No. of Cases.*	*Remaining Quiescent.*	*Retrogressing after Labour.*
Less than 1 year	17	16	1
1-2 years	34	30	4
2-3 years	10	9	1
3-4 years	19	19	—
Over 4 years	39	37	2
Total	119	111	8

One important matter is whether treatment by collapse therapy—pneumothorax, thoracoplasty or phrenic paralysis—is more efficient in protecting a patient from whatever influence pregnancy may have than conservative treatment. In this series, of the 119 quiescent, arrested, or recovered cases, 67 had been treated by collapse therapy, and 3 (4·7 per cent.) showed evidence of retrogression after labour. Fifty-two had been treated conservatively, and 5 (9·6 per cent.) of these retrogressed.

Follow-up Results

The clinical manifestations of pulmonary tuberculosis are so varied and diverse that it is hardly possible to compare control groups in the true scientific sense, and there is a tendency, perhaps natural, but scientifically unjustifiable, to convict a pregnancy as the cause of breakdown or death which may occur at any time within the next few years. Many tuberculous women, who had never become pregnant, would be dead within a comparable time. For this reason the late effects, which we will define as being after the first three months succeeding labour, are not a valid measure of the effect of pregnancy, labour, and the puerperium. Rather they would seem to be a measure of the natural processes of the disease, perhaps influenced by the associated changes produced by child-bearing in the woman's life, such as disturbed nights, the anxiety and added work of looking after an infant, and sometimes a financial strain of which the non-contributing addition to the family is the innocent cause. The author has, however, succeeded in following up 120 patients of the present series for periods varying from six months to six years, with results as indicated in the case records, and summarised in Table IV.

TABLE IV.

	Follow-up Period.	*Died.*	*Evidence of Retrogression.*	*Remaining Quiescent.*	*Became Quiescent.*
Arrested and recovered (53 cases)	6-12 months			6	
	1-2 years	1		9	
	2-3 years		2	11	
	3-4 years			5	
	Over 4 years	1		18	
Quiescent (35 cases)	6-12 months			1	
	1-2 years			9	
	2-3 years		1	7	
	3-4 years			5	
	Over 4 years			12	
Progressive (32 cases)	Up to 1 year	2*	1		2
	1-2 years	4	1	2†	5
	2-3 years	3	1		3
	3-4 years				2
	Over 4 years	1	2	1†	2

* Both deaths while still in hospital. † Unchanged or improved.

Cases followed up	120	Total cases 177.
Lost sight of	40	
Died in hospital	8	
Recent deliveries	9	

Out of 87 quiescent, arrested or recovered cases followed up, 2 (Cases 12 and 16) had died and 3 retrogressed. A total of 17 progressive cases are known to have died out of 58.

Infant Follow-up

A follow-up of the infants was carried out in 1944; 107 questionnaires were sent out and replies received in 70 cases, 11 of whom were in contact with sputum-positive mothers. Tuberculin testing was performed in only 31 of these, but 4 of the 70 infants are known to have been infected with tuberculosis. The mothers of 3 of these are known to have been infectious. Two of the 4 had died (one of miliary disease of the lungs, and one of generalised tuberculosis), one had tuberculous glands of the neck (Mantoux test positive but no indication of the type of infecting bacillus), and the fourth had enlarged mediastinal root-glands (Jelly test positive). Five other infants had died of non-tuberculous conditions.

Discussion

All tuberculosis workers have encountered cases of active, sometimes advanced, pulmonary tuberculosis who date the onset of symptoms from a pregnancy, and who did not come under expert supervision until much valuable time had been lost. Though in reported investigations—*e.g.*, Eisele and Mason (1938) and Tisdale (1938)—routine examination in ante-natal

clinics revealed a very low incidence of active tuberculous disease, one cannot escape the desirability of such examinations. There is little doubt that the inclusion of a chest X-ray as a routine in ante-natal cases would prevent the tragedies which occur in undiagnosed cases who have not been protected by being placed under favourable conditions for treatment and supervision.

So far as experience at Black Notley justifies an opinion (and the Black Notley experiment is still a unique one in this country), the crux of the whole difficult question of the effect of pregnancy on pulmonary tuberculosis appears to be whether or not adequate facilities for treatment and supervision are available. In my experience the worst cases have been those in whom tuberculosis was unrecognised or neglected during pregnancy. In particular, active pulmonary tuberculosis complicated by *severe* tuberculous laryngitis is a grave danger. In such cases, signs of chronic respiratory obstruction are liable to appear, and the consequent prolonged cardiac strain, added to the cardiac strain which is imposed by even a normal pregnancy, is only too likely to result in death from cardiac failure—*e.g.*, Cases 137, 172, 177. I regard therapeutic abortion as fully justified in such cases, or if the pregnancy is too far advanced for this when the patient is first seen, Cæsarian section at term should be considered.

Changes in Diaphragmatic Level

One of the arguments most commonly employed by those who believe pregnancy to have a harmful effect on pulmonary tuberculosis is the sudden relaxation of support produced by the fall in the level of the diaphragm which is said to occur immediately after labour. Theoretically, this seems to be a logical argument, but in practice changes in diaphragmatic level during pregnancy are of very minor importance. In the first place, belief that a beneficial effect is produced by compression of the lungs from below by the enlarging uterus is contradicted by the following points:

1. Döhrn (cited by Jameson, 1935) showed that diminution of the pleural cavity is compensated for by an increase in width of the thoracic cage, so that lung capacity is not affected.

2. Regression may occur in the early months of pregnancy and improvement in the later.

3. The general use of collapse therapy and better knowledge of the effects of changes of intrapleural pressure finally disposed of this argument.

In the second place a harmful effect produced by the emptying of the uterus is unlikely because :

1. The rise of the diaphragm does not occur till the later weeks of pregnancy.

2. It is seldom more than an inch or so, and not comparable to the elevation produced therapeutically by phrenic paralysis, with or without pneumoperitoneum.

3. In many women the uterus sinks a little before labour, usually during the last two weeks of pregnancy (" lightening "), thus reducing the time during which the diaphragm is elevated, when at all.

4. Experience with pneumoperitoneum has shown that it is very difficult

to produce elevation of the unparalysed diaphragm, and the results of this therapy without preliminary phrenic paralysis are disappointing. The abdominal capacity is increased, not so much by elevating the diaphragm as by distending the abdominal wall. Hence the induction of pneumoperitoneum immediately after labour with the object of maintaining the intra-abdominal pressure is a measure of no practical value.

Changes in Tuberculin Sensitivity

A number of writers have referred to a loss of tuberculin sensitivity during pregnancy, and this has been used to support an opinion that resistance to tuberculosis is thereby reduced.

Thus Nobecourt and Paraf (cited by Jameson, 1935) stated that 15 to 20 per cent. of pregnant women lose their tuberculin sensitivity during pregnancy, though Conn (1942) could not confirm this high proportion. Of 150 pregnant women, 90 were found to be tuberculin positive, and only 2 lost their sensitivity during the last trimester. None of these patients were suffering from active pulmonary tuberculosis. Lichtenstein (1942) considered that " a mild depression of sensitivity " occurs in about one-fourth of pregnant patients with active tuberculosis (his conclusions were based on 82 cases), but that in most instances the level of reactivity is closely comparable to similar but non-pregnant cases. None of Lichtenstein's cases became completely insensitive to tuberculin.

It is accepted that a tuberculin-negative person exposed to infection has a slightly greater chance of developing the disease than a tuberculin-positive, a fact which has been borne out by experience with young women working as nurses in sanatoria, but there does not appear to be any evidence other than hypothesis to indicate that changes in tuberculin allergy during pregnancy mean lessened resistance to tuberculosis.

Discussion of Results

In considering these, it is best to discuss the early results first, and those revealed by follow-up separately. The first point which emerges is that the number showing retrogression while in hospital in all groups was hardly a higher proportion than would be expected to occur in women of similar age group, social circumstance, and extent of disease in the absence of pregnancy. Fifteen out of 58 active cases showed evidence—often slight—of retrogression. In the normal course of the disease such a proportion could be expected, and it is unjustifiable to blame the pregnancy and labour as the cause in those in the series. Moreover, out of 32 active cases who improved following labour and while still in hospital, at least 15 are known to have maintained or bettered that improvement when followed up.

How long should her medical adviser counsel a woman whose tuberculous disease is recently quiescent to avoid pregnancy? Table III revealed the surprising fact that the interval of quiescence was not a significant factor under the conditions described in this paper, and whilst one still hesitates to advise pregnancy soon after quiescence is obtained, it does not follow that

retrogression is a probable sequel should this occur. In practice the reasonable attitude would seem to be to advise recently quiescent cases to avoid pregnancy for one to two years because of the strain imposed by the care of the infant. Where pregnancy occurred within this time, however, the outlook was still favourable so long as careful supervision was maintained—*e.g.*, Cases 50, 54, 55, 58, 64, 74, 127, 128. Patients who were treated by collapse therapy seemed to have a slightly better prognosis than those in whom quiescence was secured by conservative measures.

When followed up, the proportion of quiescent, arrested, or recovered cases retrogressing was still comparable (5 out of 88) to the earlier results (5 out of 75). When the follow-up of the progressive cases is examined in detail it is seen that all the 7 progressive cases who died in hospital and 3 (Cases 78, 133, 134) of the 10 fatal cases discovered by follow-up were advanced cases on admission whose prognosis irrespective of pregnancy was of the gravest. Moreover, 5 (Cases 80, 91, 95, 99, 136) of the remaining 7 fatal cases in this group survived for periods from nineteen months to five and a half years. Here again there was no evidence that pregnancy was a significant factor in hastening their deaths. In only one (Case 87) did a rapid post-natal breakdown lead to early fatality.

The Use of Therapeutic Abortion

Apart from cases where religious doctrine dictates a course of inactivity, the advisability of terminating a tuberculous woman's pregnancy will have to be considered in a proportion of cases. Jameson (1935) suggested that in cases of active tuberculosis decision should be delayed till the third month. If the lesion is such that the prognosis would be good if the patient were not pregnant, she should be allowed to continue her pregnancy. *Provided proper supervision and treatment can be supplied*, and that each case be considered individually, I am fully in agreement with this policy. Modern tuberculosis therapy has so changed the prognosis of these cases that many of the earlier reports, though compiled carefully and conscientiously, are no longer representative of the anticipated progress of an individual patient.

With advanced cases other factors arise, but the chances of the pregnancy going to term and a healthy baby obtained are good, and prolongation of the patient's life by abortion cannot by any means be guaranteed. In these cases, I believe that the wishes of the patient and her family can with justification be allowed to exert an influence on the final decision. As already mentioned, severe tuberculous larnygitis complicating advanced disease should be regarded as an indication for abortion, or if the pregnancy is too far advanced, for Cæsarian section at term.

In the intermediate cases of active disease, where complete quiescence is not expected, the decision is most difficult. Here an individualistic attitude must needs be taken with each case, judging upon social circumstances, parity, reaction to previous pregnancies, and the intelligence and co-operability of the patient. The danger of infection of the infant with a sputum-positive mother can be much reduced with adequate care, but without these precautions (and they are admittedly irksome and resented by many

women) the risk remains a very real one. The follow-up of infants born at Black Notley showed that 4 out of 11 who had infectious mothers became infected themselves, 2 of them fatally.

When the pulmonary disease is quiescent or arrested and the woman is adequately supervised and placed under favourable conditions, therapeutic abortion is not justified.

The Use of Contraception in Pulmonary Tuberculosis

Whether pregnancy has a deleterious effect or not upon active pulmonary tuberculosis it is a complication of treatment which it is more convenient to avoid until quiescence is attained. Patients with "untreatable" disease should be advised against pregnancy altogether. If there is no religious contra-indication, such cases, and probably also recently quiescent ones, should receive instruction in contraceptive methods, either from their general practitioner, or at the special clinics now provided by many local authorities.

Conclusions

The recent literature on the influence of pregnancy on pulmonary tuberculosis is reviewed and the differences of opinion, especially on the conduct of such cases, are again emphasised. An experience of 177 consecutive pregnancies in tuberculous patients at a maternity unit of a sanatorium is described, including a follow-up of 120. Statistical significance is not claimed for the analysis of the results; but the series is, at any rate, large enough to form a guide to the management of patients of similar clinical state and social standing, supervised in the same way.

With these qualifications, the results appear to show that pregnancy and labour rarely harm the pulmonary disease. In the great majority of these tuberculous women pregnancy was little more than an important incident in the course of their illness. Quiescent, arrested or recovered pulmonary tuberculosis ran slight risk of harm, and for these patients therapeutic abortion would not have been justified. When the pulmonary disease was active, abortion was justified only if with proper treatment and supervision favourable progress could not have been anticipated. The crucial circumstance that decided whether pregnancy would have a harmful effect or not was whether the pulmonary disease could be controlled.

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APPENDIX

CASE RECORDS

(The interval in brackets in the last column but one indicates the length of time the patient remained in hospital after labour, and that in the last column the time between labour and the follow-up examination.)

1. ARRESTED AND RECOVERED CASES

Case No.	*Age.*	*Group.*	*Clinical History before Labour.*	*Para.*	*Labour.*	*Baby.*	*Clinical State after Labour.*	*Follow-up.*
1	30	+ 2	R.A.P. 4½ years. Allowed to terminate after labour	1	2.2.40. Normal	8½ lb.	No change (4 weeks)	Disease arrested. (5 years 2 months.)
2	22	+2	R.A.P. Jan. 1937 (London Chest Hospital)	0	17.6.39. Manual rotation and forceps for P.O.P.	6½ lb.	No change (9 weeks)	R.A.P. maintained, disease quiescent. (6 years.)
3	25	+2	L.A.P. April 1933, terminated by effusion 13 months later. T.B.—since July 1934	0	11.4.39. G.A. for perineal repair	6 lb. 5 oz.	No change (9 weeks)	No change up to Dec. 1939. Thence lost sight of. (8 months.)
4	31	—	L.A.P. July 1935-Nov. 1936	1	20.9.39. Normal	8 lb. 12 oz.	No change (9 weeks)	June 1944: "Scars and calcareous deposits both upper and mid zones." "Recovered." (4 years 10 months.)
5	26	+2	9 years' history, including 8 years at Papworth to Dec. 1938; 2 courses of sanocrysin	0	19.6.39. Forceps for uterine inertia	6 lb. 11 oz.	No change (10 weeks)	Lost sight of.
6	27	+1	L.A.P. June 1936 for large lower lobe cavity. Adhesions cut Sept. 1936. Previous confinement in Black Notley Aug. 1938	1	3.7.40. Normal	7 lb. 8 oz.	No change (8 weeks)	Disease arrested. (4 years 2 months.)
7	23	—	R. pleurisy and effusion April 1935. Pleural thickening and minimal apical scarring	0	22.4.39. Normal (medical induction-disproportion)	8 lb. 14 oz.	No change (8 weeks)	Confined again (B. N.) 8.11.42. Disease arrested. (2 years 9 months.)

Case No.	*Age.*	*Group.*	*Clinical History before Labour.*	*Para.*	*Labour.*	*Baby.*	*Clinical State after Labour.*	*Follow-up.*
8	20	+1	L.A.P. for L. hilar cavity Oct. 1934	0	16.8.38. Low forceps for incomplete flexion	9 lb.	No change (9 weeks)	Contraction and fibrosis L. apex, scarring R. apex and mid-zone. (7 years.)
9	23	+2	R.A.P. March 1937 for sluggish disease of upper lobe	0	24.9.40. Normal	6 lb. 2 oz.	No change (10 weeks)	Lost sight of.
10	26	—	Sanatorium treatment Jan-Dec. 1936 for sluggish disease of R. upper lobe with small cavities	0	15.10.40. Normal	9 lb. 4 oz.	No change (10 weeks)	Classified " recovered." (4 years 1 month.)
11	31	+2	L.A.P. Jan. 1930 terminated by pleural effusion after 18 months. One course of sanocrysin in 1934. Therapeutic abortion in 1935	2	5.6.40. Normal	6½ lb.	No change (8 weeks)	Disease quiescent. (4 years 8 months.)
12	27	+2	History since 1930; in Royal National San., Ventnor, for 6 months, apparently quiescent since. Bilateral scarring of upper lobes	0	4.1.41. Normal (premature)	6 lb.	R. pleural effusion and spread of active disease in both lungs (12 weeks)	Progressive deterioration in both lungs. Died 29.8.42. (1 year 7 months.)
13	25	+2	History since 1937. Dagenham San. for 5 months, apparently quiescent since	0	20.3.41. Normal	7 lb.	No change (7 weeks)	Lost sight of.
14	34	—	Onset April 1937; minimal L. apical disease. Black Notley June-Sept. 1937	0	18.2.41.	7 lb.	No change (9 weeks)	May 1944: slight extension of focus in L. lung; no change since. Disease quiescent. (4 years 6 months.)
15	26	+2	Onset Dec. 1935. L.A.P. March 1936 (T.B.+). Ventnor July-Dec. 1936, well since	0	28.7.39.	6 lb. 12 oz.	L.A.P. tending to obliterate, but no sign of active disease (5 weeks)	Confined again in Black Notley 4.10.42. Disease arrested. (3 years 3 months.)
16	34	+2	R.A.P. Jan. 1937. Therapeutic abortion Aug. 1938. Pleural effusion and space obliterating	4	1.4.39. Normal	5 lb. 12 oz.	No change (9 weeks)	Re-admitted April, 1941. Residual encysted effusion on R., apical cavity on L. Discharged herself Nov. 1941. Thence progressive deterioration, esp. L. lung. Died 21.11.43. (4 years 7 months.)

								(4 years 7 months.)
17	32	+2	R. phrenic avulsion for soft lower lobe cavity Feb. 1935 (Black Notley)	0	20.3.41. Normal	6 lb.	Sluggish infiltration R. mid-zone 4 weeks after labour, but cleared 4 months later	Confined again Sept. 1944 (at home). Disease arrested. (4 years 5 months.)
18	35	+1	History since May 1935. Then T.B.+ without other evidence of disease. Faint shadow in R. apex on re-admission	0	15.4.41. Normal	8 lb. 10 oz.	No change (5 weeks)	Classified "recovered." (1 year 8 months.)
19	39	+2	L.A.P. in 1933, maintained for 4½ years	4	18.4.41. Normal	6 lb. 4 oz.	No change (4 weeks)	Disease arrested. (5 years 2 months.)
20	30	+2	R.A.P. Aug. 1933-April 1933 for extensive disease with cavitation. Terminated by effusion (3 air replacements)	0	19.4.39. Low forceps	6½ lb.	No change (6 weeks)	"Keeping well and working." Not X-rayed, but condition believed to be quiescent. (5 years 5 months.)
21	33	—	Pinewood San. for 4 months in 1930. One child died aged 4 months in 1938 of tuberculous meningitis	2	6.7.39. Normal	7 lb. 2 oz.	No change (4 weeks)	Lost sight of.
22	33	+2	R. phrenic avulsion Oct. 1933 for large lower lobe cavity. Last positive Dec. 1933. Cavity closed by Feb. 1934	0	28.5.41. Surgical induction for pre-eclamptic toxæmia	Still-born	No change (2 weeks)	Scarring, calcifications and contraction R. upper and mid zones. Disease arrested. (4 years 2 months.)
23	24	+2	L.A.P. Oct. 1934 for L. hilar cavity. Previously confined in Black Notley Aug. 1938 without effect on lungs and A.P. terminated soon after	1	8.1.42. Normal	8 lb. 2 oz.	No change (4 weeks)	"Recovered." (2 years 6 months.)
24	24	+1	Bilateral exudative disease in Aug. 1934 (Black Notley). R.A.P. Oct. 1934, also gold. Disch. quiescent April 1936	0	19.3.42. Normal	6 lb. 12 oz.	No change (4 weeks)	R.A.P. stopped July 1944. Sept. 1944, disease arrested. (2 years 6 months.)
25	27	—	5 years' history. Onset with L. pleural effusion and infiltration both apices	0	12.7.42. Manual rotation and forceps for P.O.P.	8 lb.	No change (5 weeks)	Disease quiescent. (3 years.)

Case No.	*Age.*	*Group.*	*Clinical History before Labour.*	*Para.*	*Labour.*	*Baby.*	*Clinical State after Labour.*	*Fellow-up.*
26	23	+1	L.A.P. Oct. 1939 (London Chest Hospital Oct. 1939-Jan. 1940)	0	28.7.42. Forceps for delay due to swollen ant. lip of cervix	7½ lb.	No change (3 weeks)	Disease arrested. (2 years.)
27	22	+2	Black Notley April 1938-July 1939. Sanocrysin and R. phrenic avulsion for bilateral exudative disease and cavity R. mid-zone	0	20.4.42. Normal	5 lb. 4 oz.	Small fresh focus L. base at 4 weeks, clearing later (8 weeks)	Confined again 6.10.43. Remained quiescent since discharge. Disease arrested. (3 years 3 months.)
28	35	+2	History since 1935. Heavy scarring and calcifications both upper lobes. Tomograms revealed small cavities buried in scar tissue	0	23.8.42. Normal	7 lb.	No change (4 weeks)	Fibro-cavernous disease R. upper zone, possible cavity L. apex. Slight deterioration; chronic disease. (2 years.)
29	20	+2	R.A.P. July 1938. Adhesions cut Feb. 1939	0	13.3.42. Normal	7½ lb.	No change (4 weeks)	Lost sight of.
30	29	+2	L.A.P. 1933. Abandoned after 4½ years; well since	0	23.2.42. Normal	7½ lb.	No change (6 weeks)	Disease arrested; thickened L. pleura only. (2 years 8 months.)
31	20	+2	L.A.P. 1935, terminated by effusion after 2 years. L. phrenic avulsion 1938	0	7.10.41. Normal	8 lb. 4 oz.	No change (10 weeks)	Confined again (B.N.) 23.2.44. Disease arrested. (2 years 5 months.)
32	30	+1	R.A.P. (Pinewood San.) June 1936. Adhesions cut Sept. 1936. Well since	0	21.8.41. Normal	5 lb. 8 oz.	No change (4 weeks)	Pleural thickening with calcification right lower zone. Contraction of lung. Disease quiescent. (4 years.)
33	26	—	R.A.P. (Black Notley) Aug. 1935. Well since. Sluggish disease of upper lobe at onset	0	11.7.41. Forceps for long 2nd stage	8 lb. 3 oz.	No change (4 weeks)	Disease arrested. (3 years 7 months.)
34	29	+2	Black Notley Sept. 1931-July 1933. R. phrenic avulsion March 1932 and L.A.P. April 1932 for exudative disease with soft cavities. L.A.P. obliterated after 14 months. Therapeutic abortion 1936	0	25.7.40. Normal	7 lb. 12 oz.	No change (7 weeks)	Disease arrested. (5 years.)
			Black Notley Jan.-May 1938 for slug-	2	27.7.41.	9 lb.	No change	Disease arrested.

35	29	—	Black Notley Jan.-May 1938 for sluggish R. sub-apical disease, sputum negative	2	27.7.41. Normal	9 lb. 2 oz.	No change (6 weeks)	Disease arrested. (4 years.)
36	34	—	Brompton Hosp. for 2 weeks in 1933, thence Pinewood San. for 17 months. R. apical scarring on admission	1	13.11.41.	8 lb.	No change (4 weeks)	Lost sight of.
37	24	—	Black Notley Jan.-June 1938, sluggish bilateral disease, discharged quiescent	0	23.7.41. Normal	7½ lb.	No change (7 weeks)	X-ray showed no change since leaving Black Notley. Disease probably quiescent. (4 years.)
38	41	—	Hæmoptysis while pregnant in 1936. Brompton Hosp. for 3 weeks, thence Pinewood San. for 3 months. R. apical scarring on admission	4	14.7.41. Normal	8½ lb.	No change (6 weeks)	Disease arrested. (3 years.)
39	22	+2	Black Notley April 1938-Aug. 1939. R.A.P. for exudative disease with cavity, terminated after 15 months by effusion	0	21.10.40. Normal	7 lb. 6 oz.	No change (4 weeks)	Disease arrested. (4 years 3 months.)
40	29	+2	L.A.P. March 1936-Sept. 1939. Second pregnancy in Black Notley (see Case 15)	1	4.10.42. Breech with extended leg. General anæsthetic and manipulation	7 lb. 7 oz.	No change (6 weeks)	Classified "recovered." (1 year 5 months.)
41	26	+2	Black Notley April-Sept. 1938 for tuberculous bronchiectasis of R. middle lobe (T.B.+). Quiescent and T.B. – since March 1940	0	28.9.42. Normal	8 lb. 2 oz.	No change (5 weeks)	Disease quiescent. (2 years 10 months.)
42	31	+2	L.A.P. March-Nov. 1935, terminated by effusion. Miscarriage 1936, stillbirth 1939. Bilateral calcified foci; found to be a severe diabetic	2	26.11.42. Normal, but intra-uterine death of fœtus suspected on 23.11.42. Still-born	8½ lb.	No change (6 weeks)	Re-admitted 25.9.44 for relapse of diabetes, cavity in L. upper lobe, bronchiectatic changes L. lower lobe. Gen. condition good, but not suitable for major thoracic surgery. (2 years 4 months.)
43	28	+2	R.A.P. May 1939 for extensive disease of R. lung with cavity (Black Notley). Discharged quiescent Feb. 1940. A diabetic	0	3.8.42. Forceps for contracted pelvis	7 lb. 2 oz.	No change (2 weeks)	Contraction of R. lung with much pleural thickening and fibrosis. Disease arrested. (2 years 11 months.)

Case No.	*Age.*	*Group.*	*Clinical History before Labour.*	*Para.*	*Labour.*	*Baby.*	*Clinical State after Labour.*	*Follow-up.*
44	22	+2	Black Notley Dec. 1936-July 1937 (R.A.P. Sept. 1936). Discharged quiescent	0	24.12.42. P.O.P., delivered face to pubis	8 lb. 3 oz.	No change (6 weeks)	R.A.P. terminated Feb. 1943. X-ray: calcified glands R. hilum and elevation of diaphragm. Disease arrested. (2 years 8 months.)
45	29	+2	L.A.P. Feb. 1936 (Black Notley) for soft cavity. Discharged quiescent Aug. 1936. Excision of tuberculous knee-joint July 1938	1	12.3.43. Normal	7 lb. 14 oz.	No change (5 weeks)	Confined again March 1945 (normal). X-ray May 1945. Heavy scarring and calcifications of both lungs and contraction of left. Disease arrested. (2 years 2 months.)
46	26	+2	Black Notley April 1931-July 1932. Sanocrysin for bilateral exudative disease. Re-admitted Jan.-Dec. 1934. Last T.B.+ March 1940	0	8.8.40. Low forceps for incomplete flexion	7 lb. 10 oz.	No change (2 weeks)	Lost sight of.
101	35	—	R. Pleural effusion April 1940. Ad. Black Notley 1.7.43. Bilateral apical scarring and residual pleural thickening on R.	3	28.7.43. Normal	8 lb. 8 oz.	No change (5 weeks)	No evidence of active disease. Disease arrested. (2 years.)
102	29	+2	R.A.P. June 1938 for acute disease R. upper lobe (Black Notley). Also tub. larynx. Disch. quiescent March 1939	0	29.8.43. High forceps for uterine inertia	7 lb. 11 oz.	No change (5 weeks)	R.A.P. terminated Nov. 1944. Contraction of R. lung with thickened pleura. No evidence of active disease. Classified " recovered." (2 years.)
103	23	+1	L.A.P. Feb. 1940, terminated by effusion May 1940 (Papworth). Heavy pleural thickening	0	4.10.43. Normal	7 lb. 12 oz.	No change (4 weeks)	Lost sight of.
104	32	+2	R.A.P. Sept. 1939 (Black Notley) for exudative disease with cavity R. upper lobe. Abandoned Feb. 1940. 7 rib thoracoplasty May 1940	0	7.6.43. Forceps for contracted pelvis	7 lb. 3 oz.	No change (6 weeks)	Disease arrested. (1 year 7 months.)
105	23	—	Bilateral exudative disease Jan. 1938. L.A.P. for soft cavity July 1938 (Black Notley). Adhesions cut Nov. 1938. L.A.P. terminated May 1942. On re-admission, scarring and contraction of L. upper lobe	0	25.12.43. Normal	7 lb. 8 oz.	No change (5 weeks)	Contraction and fibrosis L. lung. Disease arrested. (1 year 7 months.)

106	23	+2	L.A.P. Nov. 1939 (London Chest Hospital). Black Notley Feb.-June 1940. Re-admitted 28.12.44. Epileptic. L.A.P. maintained	0	27.2.45. High forceps for primary uterine inertia	7 lb. 14 oz.	No change (5 weeks)	L.A.P. maintained. Disease quiescent. (6 months.)
107	30	—	Pinewood San. in 1939 for 10 weeks. Well since. Heavy scarring and calcifications of both upper lobes	0	28.2.45. Normal	7 lb. 7 oz.	No change (5 weeks)	Lost sight of.
108	31	+2	R.A.P. Jan. 1935 for cavity in upper lobe. Disch. Aug. 1935. Confined 20.3.38 (in Black Notley). R.A.P. terminated 1938. Scarring and contraction R. lung	1	19.2.44. Normal	7 lb. 10 oz.	No change (3 weeks)	Gross contraction R. lung. Disease arrested. (1 year 5 months.)
109	34	—	Merivale San. for 6 months in 1926. Well since. Heavy scarring with calcifications in both apices	1	20.1.44. P.O.P. Manual rotation and forceps		No change (4 weeks)	Keeping well. No symptoms or P.S. (Not X-rayed.) Disease arrested. (1 year 3 months.)
110	26	+2	L.A.P. April 1939 (London Chest Hosp.). Black Notley July-Sept. 1939. A.P. obliterating on re-admission	0	26.12.43. Normal	8 lb. 7 oz.	No change (5 weeks)	A.P. obliterated shortly after delivery. Disease quiescent. (1 year 8 months.)
111	22	—	Erythema nodosum July 1937. R. pleural effusion March 1938 and some mid-zone deposit. Black Notley March 1938-Jan. 1939. Re-admitted 13.7.44; quiescent scarring R. mid-zone. Secondary anæmia	0	16.9.44. Normal. G.A. for 3rd degree tear	10 lb. 7 oz.	No change (5 weeks)	Scarring and calcifications only. Disease arrested. (11 months.)
112	22	+2	L.A.P. in 1935, terminated after 2 years by effusion. L. phrenic avulsion in 1938 (Grove Park Hosp.). Black Notley Sept.-Dec. 1941 for confinement, without effect on quiescent scarring. Re-admitted 31.12.43, still quiescent	1	23.2.44. Normal	9 lb. 3 oz.	No change (4 weeks)	Lost sight of.
113	30	—	L. pleurisy 1936. L.A.P. March 1939 (Branston San.). Thoracoscopy March 1940. Pleural thickening and scarring of upper lobes both sides	0	11.10.44. Normal	6 lb. 5 oz.	No change (3 weeks)	Lost sight of.

Case No.	Age.	Group.	Clinical History before Labour.	Para.	Labour.	Baby.	Clinical State after Labour.	Follow-up.
114	29	+2	L.A.P. Nov. 1941, obliterated after 2 years. 4.12.44, thickened pleura only	0	14.1.45. Normal	7 lb. 5 oz.	No change (2 weeks)	Lost sight of.
115	24	+2	L.A.P. July 1939 (London Chest Hosp.). Ad. Black Notley 6.11.44, good A.P.	0	4.12.44. Normal	6 lbs. 12 oz.	No change (4 weeks)	L.A.P. maintained. R. side clear. Disease arrested. (7 months.)
116	29	+1	L.A.P. 1936-39. Black Notley Feb.-May 1941. Re-admitted 2.3.44, quiescent scarring both upper lobes	0	24.3.44. Normal	7 lb.	No change (4 weeks)	Lost sight of.
117	24	—	L. pleural effusion Oct. 1940. Black Notley Feb.-April 1941. Re-admitted 16.12.43, pleural thickening only	0	8.1.44. Normal	7 lb. 6 oz.	No change (5 weeks)	"Excellent state of health." Disease arrested. (1 year 6 months.)
118	32	+2	Hæmoptysis Sept. 1940. Ad. Black Notley 26.8.43, heavy scarring of L. upper lobe	1	27.9.43. Normal	7 lb. 3 oz.	No change (6 weeks)	Disease arrested. (1 years 3 months.)
163	26	+2	Black Notley Dec. 1940-Feb. 1942. T.B.+. R. phrenic avulsion 22.2.41 (R.A.P. failed) for upper and mid-zone disease with central cavity. Discharged quiescent. Re-admitted 25.5.45, heavy scarring of R. lung, quiescent	0	7.6.45. Normal	6 lb. 11 oz.	No change (4 weeks)	
166	24	+2	R.A.P. 4.6.36. Adhesions cut July 1936 (London Chest Hosp.). Black Notley Sept. 1936-Jan. 1938. Re-ad. 22.1.44. R.A.P. maintained	0	1.4.45. Normal	8 lb. 6 oz.	No change (4 weeks)	Disease arrested.
169	40	+2	L.A.P. 1932 (Black Notley), terminated 1934. Small cavity re-opened 1936, closed on 3·12 bed-rest. Well since. Re-admitted 26.6.45, quiescent scarring L. mid-zone	0	17.7.45. Breech with extended legs	Still-born	No change (4 weeks)	
174	38	+1	Diagnosed March 1938. Extra-pleural pneumothorax Nov. 1938. Empyema space obliterated April 1940. Well since. Ad 20.6.45, heavy clouding R. apex; no evidence of activity	0	15.7.45. Normal	8 lb. 6 oz.	No change (4 weeks)	
175	30	0	Diagnosed 1939. Pinewood San. for 10 weeks. Admitted 8.2.45, heavy	0	28.2.45. Normal	7 lb. 7 oz.	No change (5 weeks)	Disease quiescent. (7 months.)

of activity								
175	30	0	Diagnosed 1939. Pinewood San. for 10 weeks. Admitted 8.2.45, heavy scarring and calcifications both upper lobes	0	28.2.45. Normal	7 lb. 7 oz.	No change (5 weeks)	Disease quiescent. (7 months.)
			2. QUIESCENT CASES					
47	22	+1	Black Notley Nov.-Dec. 1939. Sputum positive without other evidence of disease. Again T.B.+ July 1940, but no physical or X-ray evidence of disease. ? a "carrier"	1	15.1.41. Normal	7 lb. 8 oz.	No change (4 weeks)	X-ray showed no change since leaving Black Notley. Disease arrested. (4 years.)
48	35	—	History since Dec. 1940. Bilateral apical scarring on admission	5	12.4.41. Twins	7 lb. 2 oz. and 6 lb. 6 oz.	No change (4 weeks)	Re-admitted 2.11.43 with cavity right hilum, and exudative disease R. upper and mid zones. Pneumoperitoneum induced 13.12.43, discharged quiescent 11.8.44 and attends as O.P. for refills. Disease quiescent. (5 years.)
49	24	+1	L.A.P. June 1936 (Black Notley) for large lower lobe cavity. Adhesions cut Sept. 1936 (also confined Black Notley July 1940, Case 6)	0	9.8.38. Normal	8 lb.	No change (4 weeks)	Confined again July 1940. Disease arrested. (1 year 11 months.)
50	19	—	L.A.P. May 1938 (Victoria Park Hosp.). Adhesions cut Aug. 1938 (Black Notley)	0	2.2.39. Normal	8 lb. 7 oz.	No change (11 weeks)	Disease arrested. (1 year 5 months.)
51	23	+2	Complete L. thoracoplasty April 1938 (St. Mary Abbott's Hosp.)	0	17.3.40. Normal	8 lb.	No change (8 weeks)	Confined again 2.2.44 (not in Black Notley); reported "uneventful." Disease arrested. (4 years 8 months.)
52	24	—	History since Oct. 1936. Scarring of L. upper lobe on admission	0	28.12.38. Normal	7 lb. 12 oz.	No change (12 weeks)	Re-admitted 5.11.41; sluggish disease of L. upper lobe and small cavity R. subapex. L.A.P. Nov. 1941; adhesions cut March 1942. Discharged improved but not quiescent 13.6.43. L.A.P. satis.; no further change Dec. 1944. (6 years.)
53	23	—	Black Notley for 8 months in 1935, thence Merivale San. for 8 months. Heavy scarring of both upper lobes	0	7.7.38. Normal	7 lb.	Slight increase of shadows in L. upper lobe (11 weeks)	Disease quiescent. (1 year 6 months.)

Case No.	*Age.*	*Group.*	*Clinical History before Labour.*	*Para.*	*Labour.*	*Baby.*	*Clinical State after Labour.*	*Follow-up.*
54	18	—	Onset Nov. 1936, minimal R. apical disease. Scarring only on admission	0	2.7.38. Normal	9 lb. 12 oz.	No change (3 weeks)	Confined again 1940 and 1942 (not in Black Notley). Keeping well. Classified "recovered." (6 years.)
55	24	+2	R.A.P. and phrenic crush Sept. 1936 (Victoria Park Hosp.). Adhesions cut (Black Notley) March 1937. Air replacement Jan. 1938, clear fluid. On re-admission, a sterile tuberculous empyema, left lung clear	0	30.9.38. Normal	6 lb.	No change (8 weeks)	Quiescent. (4 years 7 months.)
56	26	+2	History since June 1934. R.A.P. Jan. 1935 (Black Notley) for R. mid-zone cavities. Discharged quiescent Aug. 1935	0	20.3.38. Normal	7 lb. 14 oz.	No change (9 weeks)	Confined again in Black Notley Feb. 1944. Disease arrested. (5 years 11 months.)
57	29	—	R.A.P. March 1938 (Black Notley) for small soft cavity. Terminated by obliteration after 2 years. Scarring on re-admission	0	12.10.40. P.O.P. Manual rotation and forceps	7 lb. 14 oz.	No change (8 weeks)	Disease arrested. (4 years 4 months.)
58	32	+1	Onset in 1936. In Royal National San., Bournemouth, for 6 months. Last T.B.+ March 1937	1	3.11.38. Normal	7 lb. 4 oz.	No change (4 weeks)	Disease arrested. (4 years 8 months.)
59	35	+2	R.A.P. Aug. 1938 (Black Notley) for large mid-zone cavity. Discharged quiescent Jan. 1939. (A.P. obliterated after 4 months.)	4	24.5.40. Normal	7 lb.	No change (8 weeks)	Fine scarring and calcification R. sub-apex. Disease arrested. (5 years 2 months.)
60	27	+2	Black Notley July 1938-Nov. 1939: exudative disease with cavities. Discharged quiescent	0	5.12.40. Normal	7 lb.	No change (8 weeks)	Lost sight of.
61	30	+2	L.A.P. Aug. 1939 (Gloucester Sanatorium) for small cavity L. upper lobe	0	13.1.41. Normal	9 lb. 3 oz.	No change (8 weeks)	Lost sight of.
62	26	+1	T.B.+ once before admission. Minimal stippling R. lower lobe	2	7.1.41. Normal	7 lb.	No change (5 weeks)	Disease quiescent. (1 year 4 months.)

63	32	—	L. pleural effusion Feb. 1941. Pleural thickening only on admission	1	1.5.41. Twins	Both 7½ lb.	No change (4 weeks)	X-ray within normal limits. Disease arrested. (4 years 2 months.)
64	22	+2	L.A.P. (Victoria Park Hosp.) April 1938. Obliterated April 1939. Scarring L. apex and R. upper lobe on admission	0	15.4.41. Normal	8 lb. 2 oz.	Small soft cavity formed in R. apex 5 months after labour. Closed after R.A.P. Sept. 1941 (1 year 2 months)	R.A.P. obliterated May 1944, but has kept well since discharge. X-ray July 1945: scarring of both lungs, pleural thickening on R.; no evidence of activity. Disease arrested. (4 years 3 months.)
65	26	+1	L.A.P. and L. phrenic avulsion in 1935. L.A.P. terminated July 1940. Heavy scarring L. lung on admission	0	29.1.42. Normal	6½ lb.	No change (4 weeks)	Calcifications L. upper lobe, contraction of L. lung. Disease arrested. (3 years 6 months.)
66	26	—	R. Pleural effusion April 1941	0	17.10.41. Normal	7½ lb.	No change (5 weeks)	Disease arrested. ? pregnant again. (3 years 6 months.)
67	23	—	R. apical scarring	0	7.12.41. Forceps for incomplete flexion	8¼ lb.	No change (5 weeks)	"Keeping well—quiescent." (3 years 6 months.)
68	22	—	Black Notley May-Oct. 1938 for acute exudative disease of R. lung and L. lower lobe. Rapid clearing but subsequent development of small round focus in L. apex (Sept. 1938)	0	8.8.42. Normal	8 lb.	No change (6 weeks)	Lost sight of.
69	25	—	History since 1936. Therapeutic abortion 1937. R.A.P. Oct. 1938 for apical cavity, obliterated after 2 years	0	18.10.42. Normal	9 lb.	No change (7 weeks)	Scarring R. apex. Disease arrested. (2 years 8 months.)
70	22	+1	L.A.P. (Black Notley) Sept. 1939 for cavity L. lower lobe	0	28.9.41. Breech. G.A. for manipulation	7½ lb.	No change (12 weeks)	L.A.P. obliterating. Disease quiescent. (3 years 2 months.)

Case No.	*Age.*	*Group.*	*Clinical History before Labour.*	*Para.*	*Labour.*	*Baby.*	*Clinical State after Labour.*	*Follow-up.*
71	25	+1	History since Jan. 1938. Lenham San. March 1838 for 5½ months, well since. Bilateral apical scarring on admission	0	5.1.40. P.O.P. failed, forceps, Bandl's ring, Cæsarian section. Puerperal pyrexia (*B. coli* and Staphylococci)	7 lb. 6 oz.	No change (6 weeks)	Disease arrested. (3 years 9 months.)
72	25	—	L. pleurisy Nov. 1839. Merivale San. June 1940-Jan. 1941. Heavy scarring of both apices on admission	0	4.11.42. P.O.P., manual rotation and forceps	6 lb. 12 oz.	No change (9 weeks)	Lost sight of.
73	27	—	R. pleural effusion March 1942. Black Notley May-Sept. 1942. Heavy pleural thickening on re-admission	0	7.1.43. Primary uterine inertia. Manual dilatation of cervix and rotation, and forceps	8¾ lb.	No change (9 weeks)	Disease arrested. X-ray, thickened pleura only. (2 years 7 months.)
74	30	+2	R.A.P. Aug. 1940 (Black Notley) for large cavity apex of lower lobe. Adhesions cut April and June 1941. Also infiltration L. mid-zone. Discharged quiescent Jan. 1942	0	20.10.42. Low forceps for long 2nd stage	7 lb.	No change (4½ months)	R.A.P. maintained. L. side remains quiescent. Disease arrested. (2 years 9 months.)
75	21	+2	L.A.P. June 1940 (Black Notley) for exudative disease with cavity. Also infiltration R. apex. Discharged quiescent Jan. 1941. (Therapeutic abortion March 1940.)	0	22.12.42. Normal	8 lb.	No change (3 months)	Small cavity formed in R. apex June 1943; refused re-admission. Pregnancy terminated at 4 months Feb. 1944. L.A.P. terminated by obliteration Jan. 1945; disease quiescent. R. side not quiescent, but improved. (2 years 9 months.)

119	20	—	R. pleural effusion March 1944. No aspiration. Heavy pleural thickening. (Sept. 1944)	0	21.10.44. Spontaneous delivery at 38 weeks	Still-born	Died (?) tub. peritonitis 21.1.45. Lungs: slight residual pleural thickening only	
120	27	—	L. pleural effusion Jan. 1944. One aspiration 4 oz. Residual pleural thickening and sub-apical scarring	0	25.7.44. Normal	7 lb.	No change (5 weeks)	Minimal blocking of L. pleural angle only. Disease quiescent. (1 year.)
121	23	—	L. pleural effusion Oct. 1942. Residual pleural thickening on left	1	15.2.44. Normal	6 lb. 12 oz.	No change (3 weeks)	Keeping well. X-ray, no change. (1 year.)
122	26	—	L.A.P. 25.3.43, adhesions cut Aug. 1943 (Grove Park). Admitted Black Notley 12.11.44	0	24.11.44. Normal	6 lb. 12 oz.	No change (3 weeks)	Lost sight of.
123	19	—	L. pleural effusion Aug. 1943. Admitted Black Notley 11.12.44, residual pleural thickening only	0	18.1.45. Normal	8 lb. 5 oz.	No change (2 weeks)	Lost sight of.
124	34	—	Black Notley Oct. 1941-Jan. 1942. Erythema nodosum and R. pleural effusion. Re-admitted 14.4.43, minimal pleural thickening and apical scarring (R.)	0	12.5.43. Normal	7 lb.	No change (4 weeks)	Slight scarring R. upper and mid zones. Disease quiescent. (2 years 2 months.)
125	28	+2	L.A.P. April 1941 for soft disease of sub-apex (Black Notley). Re-admitted 10.4.43, L.A.P., quiescent	3	20.5.43. Breech, extended legs	7. lb 4 oz.	No change (5 weeks)	R.A.P. maintained. Disease quiescent. (2 years 4 months.)
126	31	—	Scarring L. lower lobe with pleural thickening	3	29.5.43. Normal	8 lb.	No change (2 months)	Blocked L. pleural angle only. Disease arrested. (2 years 14 months.)
127	24	+1	L.A.P. June 1941 (Black Notley) for soft cavity L. apex. Discharged quiescent Dec. 1941.	0	7.9.43. Normal	8 lbs.	No change (5 weeks)	L.A.P. maintained. Disease arrested. (1 year 10 months.)
128	22	—	Black Notley March-July 1942 for sluggish disease L. upper lobe. Discharged quiescent	0	18.5.43. P.O.P., manual rotation and forceps	7 lb.	No change (2 months)	Disease arrested. (9 months.)

Case No.	*Age.*	*Group.*	*Clinical History before Labour.*	*Para.*	*Labour.*	*Baby.*	*Clinical State after Labour.*	*Follow-up.*
129	21	+2	Black Notley July 1940-May 1941. Calcifications R. middle lobe and hilum and exudative disease of L. lung. L.A.P. failed. L. phrenic avulsion Sept. 1940. Discharged improved but not quiescent. Re-admitted 10.12.42, quiescent scarring of left upper lobe. T.B. – .	0	22.2.43. Normal	7 lb. 14 oz.	No change (6 weeks)	Disease arrested. (2 years 3 months.)
130	27	—	R.A.P. Sept. 1943, adhesions cut Nov. 1943. A.P. abandoned Nov. 1944 for fluid. Admitted Black Notley 15.2.45, encysted pleural effusion	0	15.3.45. Normal	8 lb. 10 oz.	No change (3 weeks) Self-discharged	Lost sight of.
158	22	—	L. pleural effusion May 1940. Thickened pleura only	0	16.10.41. Normal	8 lb.	No change (4 weeks)	Lost sight of.
161	18	—	Black Notley April-Sept. 1939, for minimal bilateral disease. Re-admitted 9.9.43 pregnant	0	29.9.43. Normal	7 lb. 8 oz.	Slight increase R. sub-apex (4 weeks) Discharged herself	Seen as out-patient 23.2.44, progressive disease in both lungs. Re-admitted 11.1.45 with soft cavity in L. mid-zone. L.A.P. 26.2.45, adhesions cut 26.4.45. Aug. 1945, satisfactory L.A.P. R. lung quiescent. Disease quiescent. (1 year 11 months.)
162	23	—	Black Notley Feb.-May 1942. Minimal R. apical disease and found to be pregnant. Re-admitted 17.8.42	0	2.10.42. Normal	8 lb. 2 oz.	No change (2 months)	Disease quiescent. (1 year 4 months.)
164	21	—	R.A.P. 7.2.44 (Black Notley) for R. upper lobe disease. Adhesions cut 31.3.44. Discharged quiescent June 1944	0	19.4.45. Normal	7 lb. 12 oz.	Acute pyelitis in puerperium. Quiescent. No change (6 weeks)	
167	22	—	Quiescent scarring and calcification R. apex	0	10.4.45. Normal	9 lb. 4 oz.	No change (4 weeks)	
168	26	+1	L.A.P. Aug. 1943 (Black Notley) for exudative disease with calcification. Adhesions cut Jan. 1944. Re-admitted in labour 5.7.45. Good selective collapse	0	5.7.45. Normal	7 lb. 12 oz.	No change (6 weeks)	
176	28	+1	T.B.+ Sept. 1942 (Quantocks San. for 10 months). Last T.B.+ Jan. 1943. Admitted 7.5.45. Faint		17.6.45. Normal	7 lb. 15 oz.	Small fresh focus in L. sub-apex. Symptomless	

175	26	+1	T.B.+ Sept. 1942 (Quantocks San. for 10 months). Last T.B.+ Jan. 1943. Admitted 7.5.45. Faint scarring L. lower zone		17.6.45. Normal	7 lb. 15 oz.	Small fresh focus in L. sub-apex. Symptomless (4 weeks)
173	25	—	Black Notley Feb.-Oct. 1943 for sluggish bilateral apical disease. Re-admitted 29.5.45, quiescent scarring both apices	1	30.6.45. Normal	7 lb. 12 oz.	No change (4 weeks)
176	23	—	Black Notley Nov. 1942-March 1943 for minimal sluggish disease both upper lobes. Re-admitted 3.4.45, scarring only	0	15.5.45. Normal	7 lb. 13 oz.	No change (4 weeks)

3. PROGRESSIVE CASES

Case No.	*Age.*	*Group.*	*Clinical History before Labour.*	*Para.*	*Labour.*	*Baby.*	*Clinical State after Labour.*	*Follow-up.*
76	34	—	History since Feb. 1941. Sluggish bilateral disease with recent soft infiltration in R. lower zone	5	2.9.41. Normal	6 lb. 14 oz.	Some clearing of shadows on both sides (5 weeks)	Disease arrested; no symptoms. X-ray, quiescent scarring both upper lobes. (3 years 7 months.)
77	24	+2	R.A.P. July 1938, terminated by effusion. Small cavity R. upper lobe with fibrosis and pleural thickening	1	26.12.40. Low forceps for long 2nd stage	8½ lb.	No change (12 weeks)	Confined again 22.12.44. Cavity persists in R. upper lobe, but little change. (4 years 9 months.)
78	27	+2	"Giant" cavity R. upper lobe with infiltration all zones of both lungs	1	16.9.38 Normal	7 lb. 4 oz.	Slight improvement (3 months)	No marked change till April 1940, thence went rapidly downhill and died 16.11.40. (2 years 2 months.)
79	25	+2	Fibro-caseous disease with cavity L. upper lobe, known to be present 4 years previously. Minimal stippling R. mid-zone. L.A.P. Sept. 1938 (during pregnancy) abandoned as ineffective March 1939	0	21.11.38. Normal	6 lb. 12 oz.	Slight improvement (6 months)	Lost sight of.
80	23	—	Minimal R. apical disease	0	27.8.38. Normal	6 lb.	No change (6 weeks)	Re-admitted July 1939 with extensive disease of L. lung and cavity R. apex. Baby also admitted suffering pulmonary tuberculosis and died tub. meningitis Sept. 1939. Mother died 17.7.40. (1 year 11 months.)

Case No.	*Age.*	*Group.*	*Clinical History before Labour.*	*Para.*	*Labour.*	*Baby.*	*Clinical State after Labour.*	*Follow-up.*
81	30	—	Bilateral fibro-caseous disease of upper lobes. Small cavity on R. History since Jan. 1936, miscarriage March 1936	0	22.3.38. Normal	6 lb.	No change (9 weeks)	R. apical cavity smaller, fresh disease L. upper and mid zones. (5 years 5 months.)
82	25	+2	9 years' history. Gross disease of L. lung with contraction, and infiltration of R. upper and middle zones	2	—	—	5 weeks after admission died of tub. meningitis following generalised infection	
83	24	+2	Stippling in R. apex and mid-zone, extensive fibro-caseous disease of L. lung with hilar cavity	0	21.9.38. Normal	7 lb.	No change at 3 weeks. L.A.P. Oct. 1938, discharged quiescent June 1939 (9 months)	May 1942 (3 years 8 months after labour) re-admitted for small focus in R. apex which cleared on conservative treatment. Quiescent May 1945. (4 years 8 months.)
84	25	—	Large thick-walled cavity L. upper lobe, some stippling L. lower lobe	1	12.1.41. Normal	7 lb. 8 oz.	No change (8 months)	Lost sight of.
85	22	—	Sluggish bilateral disease of both upper lobes	0	1.8.39. P.O.P., manual rotation and forceps	8 lb.	Marked improvement on both sides (4 weeks)	Re-admitted, sluggishly active disease in both lungs. (4 years 10 months.)
86	28	+2	Onset Dec. 1940. Extensive bilateral disease on admission	2	19.7.41. Normal	5 lb. 3 oz.	Rapid retrogression in last weeks of pregnancy. Death 6 weeks after labour	
87	33	+2	Chronic fibro-caseous disease of both lungs with large cavity in L. upper lobe	0	2.9.41. Still-birth (30 weeks)	—	Febrile. Spread of disease in both lungs	Died 21.11.41. (2 months.)
88	30	—	L. pleural effusion and apical infiltration	0	1.6.41. Normal	7 lb. 4 oz.	Small fresh focus L. sub-apex (4 weeks). Cleared 3 months later (4 months)	Keeping well, no evidence active disease. (4 years.)
89	35	+2	R.A.P. (Black Notley) in 1937 for 2 cavities in R. upper lobe, maintained for 5 months. Pleural thickening, and infiltration R. mid-zone on re-admission	2	22.1.42. Normal	7 lb. 4 oz.	T.B.+ once only, 4 days before labour. R. mid-zone disease slightly more marked (6 weeks)	Lost sight of.

90	39	+2	History since 1938. Also mitral stenosis. Fibro-caseous disease both upper lobes, with cavity on left	4	11.3.42. Normal	3 lb. 4 oz. (5 weeks premature)	Slight improvement (3 weeks), also at 3 months (5 months)	Reported by Tuberculosis Officer Oct. 1942 "condition unchanged," but died one month later. (8 months.)
91	26	—	Fibro-caseous disease R. upper and middle lobes, some infiltration L. mid-zone	2	18.11.41. Normal	8 lb.	Slight clearing both sides (4 weeks)	Slowly progressive disease. Died 24.12.43. (2 years 1 month.)
92	26	—	Sluggish bilateral apical disease	0	9.1.39. Low forceps for long 2nd stage	7½ lb.	Marked clearing both sides (11 weeks)	Lost sight of.
93	26	+2	L.A.P. April 1837 (Victoria Park Hosp.) for exudative disease with cavity. Small cavity persisting in L. lower lobe	1	4.7.41. Normal	8¼ lb.	No change (13 weeks)	Lost sight of.
94	26	+2	Fibro-caseous disease of R. upper lobe	2	2.3.42. Normal	7 lb. 6 oz.	R. phrenic avulsion June 1942, discharged quiescent Nov. 1932 (8 months)	Lost sight of.
95	21	+2	R.A.P. March 1941 (Oldchurch County Hosp.) for broncho-pneumonic disease of whole of R. lung. After labour: R. phrenic avulsion, intravenous gold, and adhesions cut	1	21.8.41. Normal	7 lb. 2 oz.	Fresh focus L. sub-apex (4 weeks). Both sides quiescent (1 year 9 months)	L. side re-activated about Aug. 1943 and regressed rapidly. R.A.P. abandoned. Died following hæmoptysis 23.6.44. (2 years 10 months.)
96	24	+2	Gross bilateral disease, with cavity R. upper lobe. Also tuberculous enteritis	0	13.6.41. Normal	6½ lb.	Gradual recovery and discharged quiescent (1 year 10 months)	Disease quiescent. (1 year 6 months.)
97	25	—	Sluggish bilateral disease both upper lobes	0	4.11.42. P.O.P., manual rotation and forceps	6 lb. 12 oz.	No change (12 weeks)	Lost sight of.
98	22	+2	Exudative disease L. lung with large central cavity. R.A.P. attempted, no space	0	23.1.42. Normal	7½ lb.	No change (2 weeks). R. phrenic avulsion June 1942. Discharged quiescent March 1943	"Very well." Quiescent. (3 years 6 months.)
99	23	—	R. apical infiltration with pleural thickening	0	5.6.42. Normal	7 lb. 3 oz.	Fresh focus L. lung and cavity R. apex (7 weeks)	Progress reports incomplete. Died 5.2.44. (1 year 7 months.)

Case No.	*Age.*	*Group.*	*Clinical History before Labour.*	*Para.*	*Labour.*	*Baby.*	*Clinical State after Labour.*	*Follow-up.*
100	29	+2	Fibro-caseous disease of whole of R. lung with large cavity in upper lobe. R.A.P. Nov. 1942 (while pregnant)	1	4.3.43. Normal	8 lb. 11 oz.	No change (4 weeks). Adhesions cut April 1943. Quiescent (2 months)	R.A.P. maintained. Quiescent. (2 years 4 months.)
131	41	+2	L.A.P. Dec. 1937, L. phrenic avulsion Nov. 1938 (Black Notley). L.A.P. terminated Feb. 1942. Re-admitted 23.11.44, pleural thickening on L., small area of tub. infiltration R. sub-apex	1	12.12.44. Normal	7 lb. 7 oz.	No change (4 weeks)	Contraction of L. lung with pleural thickening and fibrosis of upper zone. Fibrotic inactive lesions R. sub-apex. Disease arrested. (7 months.)
132	23	—	L.A.P. 10.9.43 (Southend Hosp.). Admitted Black Notley 1.11.43	0	16.1.44. Normal	7 lb. 1 oz.	Adhesions cut 18.2.44. Improved collapse. Discharged quiescent (3 months)	Lost sight of.
133	27	+2	Advanced bilateral fibro-caseous disease	0	23.7.43. Normal	7 lb. 6 oz.	No change at 4 weeks. Improvement (6 months)	Refused supervision. Died 22.2.45. (1 year 7 months.)
134	21	+2	Gross bilateral disease of coarse miliary type with cavity in R. upper lobe. Severe tub. laryngitis	0	1.12.42. P.O.P., manual rotation and forceps	6 lb. 1 oz.	Slowly progressive disease in both lungs. Large cavities both upper zones (1 year 2 months)	Steady deterioration. Died 16.9.44. (1 year 9 months.)
135	23	—	Minimal focus R. sub-apex. Discovered as contact	0	24.7.43. Forceps for long 2nd stage. Copious vomiting	9 lb.	Aspiration broncho-pneunomia, recovery. Discharged quiescent, clearing of sub-apical focus (3 months)	Disease quiescent. (2 years 1 month.)
136	31	+2	Chronic fibro-caseous disease of R. lung with apical cavity	1	2.3.39. Normal	5 lb.	No change (4 weeks)	Died 16.9.44. No intermediate records. (5 years 6 months.)
137	36	+2	Bilateral disease of sluggish type. Very severe tub. ulceration of larynx with extreme stridor. Extension in L. lung 2 weeks before labour	0	13.4.43. Normal (premature 2 weeks)	5 lb. 8 oz.	Extreme dyspnœa due to larynx. Cardiac failure and death 21.4.45	

138	39	—	L. pleural effusion Dec. 1942. Admitted Black Notley 26.1.43. Air replacement 30 oz. fluid. Severe macrocytic anæmia of pregnancy	0	9.4.43. P.O.P., manual rotation and forceps	6 lb. 15 oz.	Residual pleural thickening only. Blood count normal (3 months)	Re-admitted 11.11.44, small L. apical cavity, T.B.+. L.A.P. failed. General condition excellent. Recommended thoracoplasty. (1 year 3 months.)
139	22	+2	Admitted 4.4.44 L. sub-apical infiltration, R. apical scarring and small right pleural effusion. Mirror test T.B.+	0	3.6.44. Induction for albuminuria and rising B.P.	6 lb.	Symptomless and T.B.–. Marked clearing of left sub-apex (4 months)	Disease quiescent. (1 year 2 months.)
140	32	+2	R. pleurisy 1937, L. pleurisy April 1944. T.B.+ 7.6.44. Admitted 10.8.44, large apical and smaller mid-zone cavity on L., bilateral pleural thickening	0	23.8.44. Normal	7 lb. 14 oz.	Both cavities slightly smaller (6 weeks). Transferred to L.C.C. San.	Lost sight of.
141	36	—	Small soft cavity R. apex. Admitted 25.6.43	2	28.7.43. Normal	7 lb. 4 oz.	Cavity closed 4 weeks after labour. Discharged quiescent (5½ months)	No change until Aug. 1945, when a little ill-defined fresh infiltration appeared in R. mid-zone. (2 years 1 month.)
142	30	+2	L. phrenic crush 1935 and avulsion 1940. T.B.+ 11.1.43. Admitted 3.7.44, small cavity L. apex embedded in heavy scar tissue (tomograph)	0	27.8.44. Normal	—	No change (10 weeks)	No change. (1 year.)
143	26	+2	R.A.P. June 1939 (Aberdeen), obliterated after 1¾ years. R. pleurisy March 1943. Admitted Black Notley 21.4.43, pleural thickening on R., small cavity L. apex. L.A.P. 23.8.43	0	25.9.43. Normal	Less than 3 lb. (2½ months premature). 9 lb. 4 oz. on discharge	Adhesions cut 31.12.43. Good collapse of lung. Discharged quiescent (4 months)	L.A.P. maintained; disease quiescent. (1 year 9 months.)
144	23	—	R.A.P. 16.1.43, adhesions cut Aug. 1944 (Harefield). Admitted Black Notley 16.10.44, R.A.P., and sluggish disease with small cavity in L. upper lobe	0	30.11.44. Normal	7 lb. 8 oz.	L. apical cavity closed 2 weeks after labour. Self-discharged (3 weeks)	Lost sight of.

Case No.	*Age.*	*Group.*	*Clinical History before Labour.*	*Para.*	*Labour.*	*Baby.*	*Clinical State after Labour.*	*Follow-up.*
145	33	+2	Extensive bilateral disease with multiple cavities	0	3.10.44. Normal	8 lb. 8 oz.	Improvement both sides 2 weeks after labour. L.A.P. 1.11.44. Died 6.12.44 acute cardiac failure	
146	22	—	R.A.P. April 1944, self-terminated. Admitted Black Notley 4.12.44. Fibro-caseous disease R. upper lobe with small cavity	1	23.12.44. Normal	5 lb.	Slight clearing of exudate (4 weeks)	Lost sight of.
147	34	+2	Bilateral exudative disease, mid-zone cavity on R. Shallow tub. ulceration of larynx	1	10.1.45. Normal	6 lb. 3 oz.	Marked clearing both sides. Cavity smaller. Larynx: residual congestion of L. cord (5 weeks)	Lost sight of.
148	24	+2	R. phrenic avulsion May 1940 (St. Andrew's Hosp.). Chronic fibro-caseous disease R. lung, T.B.+. Tub. ulceration of larynx	0	13.11.44. Normal	6 lb. 12 oz.	Increased opacity (partly atelectasis) (4 weeks)	"Looks ill. T.B.+." Increased opacity R. lung. Baby died tub. meningitis 7.4.45. (7 months.)
149	19	+2	R.A.P. 1.1.45 for exudative disease of R. mid and lower lobes. (Northern Hospital)	0	27.2.45. Normal. (Induction at 38 weeks for a slight general contraction)	5 lb. 14 oz.	Improved collapse. Gen. cond. much improved. Awaiting thoracoscopy (4 weeks)	Lost sight of.
150	28	+2	7 years' history sluggish bilateral disease. L. pleural effusion Feb. 1943	0	28.1.44. Low forceps	9 lb. 4 oz.	Slight increase of exudate on both sides. Self-discharged (4 weeks)	Lost sight of.
151	22	+2	T.B.+ May 1944. Fibro-caseous disease of L. upper lobe	0	27.5.44. Normal	9 lb. 3 oz.	No change. T.B. – (4 weeks)	Lost sight of.
152	23	—	L. pleural effusion May 1944. Considerable residual thickening and small quantity of fluid	0	23.9.44. Normal	6 lb. 10 oz.	No change (4 weeks)	Lost sight of.

153	26	+2	T.B.+ Jan. 1944. Admitted Black Notley 11.9.44, small cavity embedded in scar tissue L. apex	0	14.10.44. Normal	6 lb. 4 oz.	Cavity less obvious. Self-discharged (3 weeks)	Lost sight of.
154	24	+1	Admitted Black Notley 9.9.43, soft cavity base of L. upper lobe. L.A.P. 20.9.43, thoracoscopy 25.11.43	0	27.4.44. Normal	8 lb. 8 oz.	Good collapse of lung. Quiescent since Jan. 1944 (4 weeks)	L.A.P. maintained. Disease quiescent. (1 year 2 months.)
155	27	—	Diagnosed June 1943, bilateral pul. tub. of coarse miliary type. Admitted Black Notley 22.9.43. Serial X-rays showed progressive clearing	0	15.11.43. Normal.	7 lb. 1 oz.	Further slight clearing on both sides (15 weeks)	"No symptoms" (not X-rayed). Probably quiescent. (1 year 8 months.)
156	33	—	Symptomless; discovered by routine ante-natal X-ray in May 1944 (L.C.C.). Admitted Black Notley Aug. 1944, bilateral miliary stippling. Irregular fever commenced end of Sept. 1944; no change in X-ray	0	19.10.44. P.O.P. and large ant. lip of cervix. Manual rotation and forceps	Still-born	Fever continued at higher level. Rapid onset of fulminating tub. meningitis 24.10.44. Died in 12 hours	
157	31	—	R. pleural effusion June 1944. Heavy residual pleural thickening	0	19.8.44. Normal	6 lb. 5 oz.	No change (4 weeks) Self-discharged	Lost sight of.
159	28	+2	Brompton and East Anglian San. 1938-39, ten T.B.+ but well since. Admitted Black Notley 9.11.42. Sluggish disease of R. apex. T.B.+ 16.11.42	0	14.1.43. Normal. Breast abscess 19.1.43	7 lb. 8 oz.	Sputum negative since admission. No change in X-ray (4½ months)	Disease quiescent. (2 years 7 months.)
160	40	+2	Fibro-caseous disease L. upper and mid-zones with cavitation. Tub. laryngitis. L.A.P. 11.8.43	2	18.9.43. Normal	5 lb. 12 oz.	A.P. abandoned Sept. 1943 (adherent apex). Gen. condition improved. "Hard" cavity L. apex (4½ months)	Slight increase in disease L. lower zones, apical cavity persists, otherwise no change. (1 year 9 months.)
165	28	+1	Minimal R. sub-apical disease (T.B. –)	0	19.3.45. Normal	6 lb.	T.B.+ 26.3.45. No change in X-ray. Transferred to L.C.C. San. (R.A.P. advised)	Lost sight of.

Case No.	*Age.*	*Group.*	*Clinical History before Labour.*	*Para.*	*Labour.*	*Baby.*	*Clinical State after Labour.*	*Follow-up.*
171	42	—	Admitted 8.1.45, heavy tuberculous infiltration R. mid and upper zones, some contraction of R. lung. (Dispensary observation since May 1944, diagnosis confirmed although sputum negative)	4	8.4.45. Normal	8 lb. 13 oz.	Pyelitis in early puerperium. Progressive clearing of pulmonary shadows. Discharged quiescent 25.8.45. Scarring of right mid-zone only	"Keeping well," disease quiescent. (6 months.)
172	32	+3	Admitted 11.6.45. Bilateral miliary disease. Sever tub. laryngitis with extreme œdema and congestion. An advanced case with hopeless prognosis	3	16.7.45. Cæsarian section (spinal anæsthesia)	6 lb. 5 oz.	Progressive asthenia. Died 29.7.45	
177	26	+3	Advanced bilateral disease with cavitation. Severe tub. laryngitis with much stridor (treated for a year as "asthma.") A dying woman. Ad. 4.6.45	0	Cæsarian section proposed, but died suddenly of acute cardiac failure 6.6.45			

OBSERVATIONS ON MYOTATIC IRRITABILITY

By LAURENCE MARTIN
From Addenbrooke's Hospital, Cambridge

THE linear contraction-bands which can be produced in the pectoralis major muscles by tapping their converging fibres below the outer portions of the clavicles are termed myotatic irritability or myoidema.

The original description of this phenomenon is obscure, but it survives in modern textbooks in the chapters on pulmonary tuberculosis, although suggestions as to its nature are mainly conjectural. Thus Keers and Rigden (1945) believed that myotatic irritability was commonly found in cases of pulmonary tuberculosis, although not peculiar to it, and that it was possibly due to tuberculous toxins having rendered the muscles unduly irritable. Young and Beaumont (1941) stated that myoidema was an undue irritability of the muscles and might occur in pulmonary tuberculosis at all stages. They added, however, that it was not pathognomonic and might occur in any cachectic state.

It is clear, therefore, that myotatic irritability is certainly regarded as pathological, is associated with wasting, and is so frequently observed in pulmonary tuberculosis as only to be mentioned in connection with that disease.

The object of this note is to suggest that myotatic irritability is a normal and physiological phenomenon of the "stretch-reflex" type, comparable with the knee-jerk, and to consider the main factors influencing its perception.

PRESENT STUDY

Myotatic irritability was repeatedly observed in healthy soldiers sent for precautionary X-ray examination of the chest and in healthy nurses who were examined clinically and by chest radiography at the commencement of training. It thus appeared that the phenomenon could not be attributed to any pathological cause and that it was simply a "stretch-reflex" in which a blow upon the tendon of a muscle produced a contraction of its fibres. It could not, however, be elicited in every individual examined, so further observations and enquiries into anatomical factors were carried out.

ANATOMICAL FEATURES OF PECTORALIS MAJOR

The pectoralis major is a fan-shaped muscle whose fibres extend from their main origins on the sternum and inner portions of the clavicles to converge into a flat, fairly narrow tendon which is inserted into the neck of the humerus. The muscle is composed of bundles of fibres which remain comparatively separate during their convergence before finally merging into the tendon. Thus when the pre-tendinous part of the muscle below the outer part of the clavicle is tapped, the whole muscle does not contract, but only those bundles which are represented at the point where the blow falls.

It is thus possible to produce contraction in the upper, middle or lower bundles of the muscle by varying the position of the blow upon the pre-

tendinous convergence. In this respect the response differs from that of the quadriceps muscle in production of the knee-jerk, for that muscle, by virtue of its penniform constitution and composite tendon, responds as a whole to a blow upon the patellar tendon. Nevertheless the fundamental stretch-reflex effect is the same in both muscles in that a blow upon the tendon will elicit a brisk contraction of the associated fibres.

Effect of Subcutaneous Fat overlying the Pectoral Muscles

It appeared from observations on the healthy subjects mentioned above that subcutaneous fat overlying the pectoral muscles could mask the linear contractions and so account for the apparent absence of myotatic irritability in well-covered persons. This was particularly noticeable in women whose mammary fat may extend high up towards the axillæ and clavicles.

The following observations were therefore made on hospital patients with varying degrees of obesity, spareness of habitus, or wasting, in order to note the effect of subcutaneous fat in masking myotatic irritability. The patients examined fell into four groups (see Table); they were unselected apart from the fact that X-ray examination of their chests (dictated by the needs of the individual cases) showed no pulmonary tuberculosis or gross abnormality. Diagnoses included peptic ulcer, hypertension, asthma, bronchitis, heart disease and abdominal carcinoma.

Group.	*Number of Patients.*	*Myotatic Irritability.*	
		Observed.	*Not Observed.*
1. Obese or well covered	4	0	4
2. Average habitus, no loss of weight	6	4	2
3. Naturally thin, no loss of weight	5	5	0
4. Considerable loss of weight or emaciated ..	5	5	0
Totals	20	14	6

Two cases of active untreated pulmonary tuberculosis were also examined and myotatic irritability was observed in both.

These results show that obesity or considerable subcutaneous fat prevented perception of myotatic irritability, while a spare habitus, considerable loss of weight, or emaciation favoured it. The two subjects of group (ii.) in whom myotatic irritability was not observed were women with much mammary fat.

Conclusions

Myotatic irritability is one of those physical signs which are only sought for in special cases when their presence is anticipated. They thus acquire a spurious pathological significance because their occurrence in healthy persons is not appreciated.

This small investigation has shown that myotatic irritability occurs in normal individuals, but that its perception is governed by the amount of subcutaneous fat overlying the pectoral muscles. It is thus readily observed in persons with active pulmonary tuberculosis or other wasting diseases, but has no pathological significance in itself.

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BEAUMONT, G. E., and YOUNG, R. A.: Price's Textbook of Medicine, 1941, 6th ed., London.
KEERS, R. Y., and RIGDEN, B. G.: Pulmonary Tuberculosis, 1945, Edinburgh.

REPORTS OF SOCIETIES

JOINT TUBERCULOSIS COUNCIL

AT their meeting on November 17 the Joint Tuberculosis Council paid tribute to the late Dr. Ernest Ward. For a long period Dr. Ward was Hon. Secretary of the Council and he did much to organise it in its early days.

The chief business of the meeting was to consider a report drafted by the Council's Committee on the Development of the Tuberculosis Services. The report, which was approved with some amendments, is at present in the printer's hands and therefore cannot be given in full here, but its main points are as follows:

1. Tuberculosis work should be organised regionally, and joint boards should be set up for the purpose.
2. The joint boards should have executive powers.
3. The areas served by tuberculosis boards should be approximately the same size as the new regions under the projected National Health Service; local administrative units, however, should serve a population of about 250,000.
4. The senior clinical tuberculosis officer for each region should have access to the joint board as of right.
5. The environment and preventive aspects of tuberculosis work—*i.e.*, the dispensary and domiciliary work, should not be divorced from the clinical aspect. Tuberculosis clinicians, therefore, should have responsibility for their work at all its stages.
6. A not inconsiderable advantage of a remodelled tuberculosis service would be the opportunities for promotion which it would create, thus providing the incentive for the best clinicians to devote themselves to the work.
7. The Council reiterate their frequently expressed conviction that a *sine qua non* of a successful tuberculosis service is recognition of the tuberculosis clinician as a specialist, accorded rank and pay appropriate to his status.

At the same meeting the Joint Tuberculosis Council approved the final draft of a Memorandum of Advice on Mantoux Conversion of Hospital and Sanatorium Staffs. The main provisions of this Memorandum have been

given in a previous Press statement. Copies of the completed Memorandum (and copies of the Development Committee's report) may be obtained from the Hon. Secretary of the Council, Dr. Norman England, 1, Becket Street, Oxford.

The officers of the Joint Tuberculosis Council reported on a recent meeting with the Ministry of Health representatives, at which the Council's draft report on tuberculosis notification was discussed. There was a frank exchange of views, and the Ministry promised to give the Council's ideas the fullest consideration.

The Nursing Committee was asked to consider Pamphlet P.L. 177, forwarded by the National Advisory Council on Nurses and Midwives, which contained recommendations about the recruitment of nurses and midwives for training in institutions.

Dr. D. P. Sutherland, Senior Tuberculosis Officer, Manchester, was nominated as next year's chairman of the Council, with Drs. Peter W. Edwards and G. Jessel as vice-chairmen. Drs. A. P. Ford and Norman England were re-nominated as Hon. Treasurer and Hon. Secretary respectively.

NOTICE OF MEETING

ART THERAPY CONFERENCE, NOVEMBER 29, 1945

THERE is no doubt that a new attitude is coming over rehabilitation of tuberculous patients. No longer need we think exclusively of chicken farms, garden seats, leather bags and the routine of factory life. The tuberculous subject has very often high mental endowment, and now it appears that the field of art and creative design is one in which he can obtain constructive openings. The matter was discussed at a Conference called by the NAPT, and very largely attended by representatives of the medical profession, occupational therapists and artists. It is interesting to note that there are at least eighty-three industries in this country where draughtsmen, craftsmen, designers and workers in decorative and applied art are employed.

The chair was taken by the Duchess of Portland, who said that congenial employment was for tuberculous patients the breath of life. There was no reason why such patients should not produce first-class work of commercial value, and not merely something which depended for its sale on the charitable appeal of the sad label " Work by the Disabled."

Sir William Crawford stressed the quality of British goods, but said they were not presented attractively enough and lacked colour, form and design. Here was a field for the tuberculous patient—a field offering a continual new interest which was beneficial to the patient, and one in which the work was both light and remunerative. If the scheme could be put into practice it would benefit not only the patients, but British manufacture as well.

Mr. Philip James, Director, Council of Arts, said that we did not yet fully understand the healing powers of art. Art was life, like love and religion; it was one of those things which exist in their own right and which man finds absolutely indispensable for his own fulfilment. From the practical point of view there were two essentials: that instructors should be experts who would encourage creative design, not slavish copying, and that goods produced should

be of commercial value and not what might be described as "just Christmas presents."

Mr. H. W. Yoxall, Managing Director of *Vogue*, emphasised the importance of good design, both for selling goods and for its therapeutic effect. He felt it essential that the work taught should be work which would sell on its own merits and not on charitable appeal, and advised concentration on industrial design for which there was an assured market, rather than on arts and crafts which required specialised selling.

Dr. Fowler, Medical Superintendent of Pinewood Sanatorium, thought from his own experience that about 10 per cent. of patients had definite potentialities, but that every tuberculous patient would benefit from the raising of the general standard of culture. He referred to the question of infection, pointing out that all articles made were easily and quickly disinfected.

Many other interesting points were raised in discussion Dr. R. R. Trail, Medical Director of Papworth Village Settlement, pressed for two practical measures: the appointment of artists to teach people on an agreed scheme, and the obtaining of definite offers from commercial firms to consider the designs and work produced. He felt if these were accomplished a great step forward would have been taken. These conclusions embodied the general opinion of the meeting.

These general ideas have now to be translated into action, and the NAPT intends to make a practical scheme through which any tuberculous patient needing help, coaching and assistance in exploiting his talent to the full in arts and crafts will not fail to get it.

MEMORANDUM ON THE TEACHING OF TUBERCULOSIS TO MEDICAL STUDENTS FROM THE TUBERCULOSIS EDUCATIONAL INSTITUTE

THE following memorandum has been received from Dr. Frederick Heaf and Dr. Harley Williams. It is given verbatim without comment for the benefit of readers.

1. No one will deny that the teaching of tuberculosis to medical students is far from satisfactory. It is still only too common an experience to find that a newly qualified practitioner's only acquaintance with tuberculosis is based on physical signs of cases in advanced stages of the disease and a very theoretical knowledge of sanatorium routine. The vast field of the preventive and socio-medical side of the subject and even the importance of early diagnosis are largely unknown to him, so that he enters practice quite unable to appreciate the significance of one of the commonest and most serious diseases.

2. In the Goodenough report on medical schools it is stated that the present facilities for teaching tuberculosis vary considerably in teaching centres, and it gives two examples which indicate the differences which exist in the nature of the instruction, showing how it depends mainly on the presence or absence of a tuberculosis department or dispensary attached to the hospital of the medical school. The situation as it was in 1939 is summarised at the end of the report. At that date, of the 25 medical schools in England, Scotland and Wales, 12 provided teaching in tuberculosis but not in the main

hospitals. Of these 12, 9 made use of the treatment beds of the local authority. Four teaching hospitals made use of the facilities at special chest hospitals. Only 2 hospital medical schools had beds of their own reserved specially for tuberculosis cases. It would appear that in over 50 per cent. of the medical schools it was only by good fortune and not by any arrangement on the part of the authorities that the student was able to acquire knowledge on tuberculosis. In such schemes where it was possible for the student to take a course in tuberculosis it was usually quite optional and dependent on the personal inclination of the student himself. It not infrequently happened, therefore, that the student passed through his medical training seeing only a few cases of pulmonary tuberculosis, and if he did it would be those with advanced disease, so that the mental picture he formed of the disease would be one of crepitations, râles and pulmonary cavities, and a hazy idea that all such patients require sanatorium routine with the addition of artificial pneumothorax treatment if it could be given. It is not surprising, therefore, that he does not recognise the disease until it has reached this advanced stage.

3. A more recent survey made by the Tuberculosis Educational Institute has revealed some improvement in the situation, particularly in the northern medical schools. In nine teaching schools compulsory courses in tuberculosis are arranged and short periods of residence at the local authorities' sanatorium are included in the curriculum. A number of visits are paid to the local dispensary, so that the student begins to appreciate the socio-medical side of the problem and the value of early diagnosis. These arrangements all show how necessary it is to link up the tuberculosis services of the local authority with the medical schools for educational purposes. In this matter local authorities might use the old advertisement slogan: "You want the goods—we have them."

4. The Committee of the Tuberculosis Educational Institute has given considerable attention to the undergraduate and post-graduate facilities for studying tuberculosis in all parts of the country, and it feels that the time has come to emphasise the need for putting tuberculosis tuition in a more prominent position in the medical training of the student and to arrange, uniformly in all schools, easy access to the local authorities' tuberculosis services, so that the vast amount of material being dealt with at the many dispensaries and sanatoria in the country may be used for the teaching of tuberculosis in all its aspects.

5. The Committee strongly support the recommendation in the Goodenough report that there should be a tuberculosis dispensary at or adjoining every parent teaching hospital, and that in-patient facilities for cases of both pulmonary and non-pulmonary tuberculosis should exist in one or more of the hospitals constituting the teaching centre, provided there is a chest physician or orthopædic surgeon in charge of the beds, or a chest hospital within easy reach. It does not, however, consider that such facilities should exclude a series of visits to sanatoria from the curriculum, for it is almost impossible to convey the meaning of sanatorium treatment to a class of students around the bedside in the wards of a general hospital.

The Committee wish to amplify the scheme of the Goodenough report by suggesting that:

1. The physician in charge of the dispensary and/or the medical superintendent of the sanatorium might with advantage be appointed a lecturer on the staff of the medical school which may be situated within the region or area which the dispensary or sanatorium serves.

2. All medical students must be attached to approved tuberculosis dis-

pensaries as clinical clerks for short periods, during which time they should also become acquainted with the technique and organisation of mass radiography.

3. Facilities should be made available for short periods of residence at approved sanatoria for medical students who wish to make a further study of the subject.

It will probably be argued that the medical curriculum is already heavily loaded and it would be difficult to find time for these suggestions. The Committee are well aware of this difficulty, but would suggest that those diseases which have the highest incidence should receive priority in a teaching syllabus, even if it meant sacrificing time on the rare conditions which always have a fascination and often assume an exaggerated importance in medical education for a qualifying degree.

REVIEWS OF BOOKS

Introduction to Diseases of the Chest. By JAMES MAXWELL. 2nd ed. Hodder and Stoughton. Price 12s. 6d.

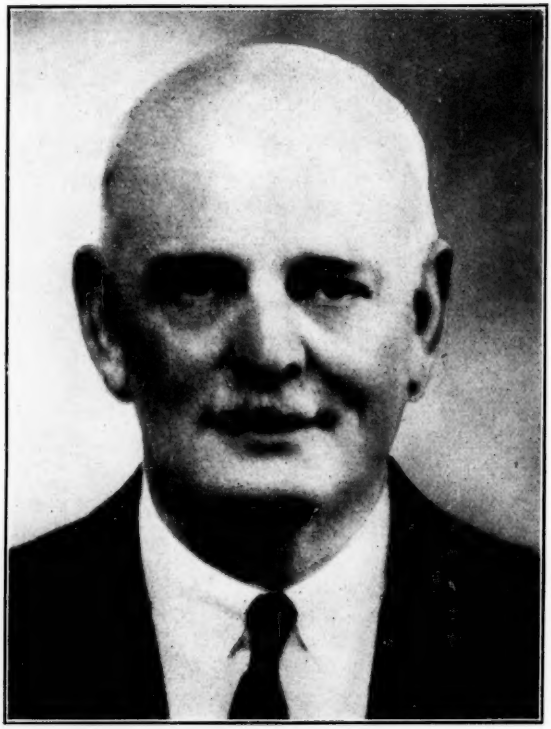
This book, as the title shows, is intended mainly for students. It is clear and concise. Stress is laid on symptomatology and physical signs, to which eight chapters are devoted, and they are emphasised throughout the book. It is a pity that the author has not found space for a description of the anatomy of the chest, and particularly of the bronchi. These are not adequately dealt with in anatomy textbooks and are surely of prime importance, particularly in relation to posturing, as the author himself emphasises. Postural treatment is too often inadequately undertaken through ignorance of its basic principles.

A short chapter on the interpretation of radiographs is excellent and to the point. But the author's description of bronchography, which is detailed, places little emphasis on positioning of the patient and possibly accounts for the quality of the normal bronchograms displayed in Figs. 7 and 8.

A further small criticism is offered. It is surely time that textbooks, particularly those intended for students, stopped advocating the exhibition of morphia in the treatment of a severe asthmatic attack, or at least gave some warning of its dangers and advice as to regulation of dosage.

In the main, however, this is a good book for its purpose and can be recommended.

PLATE I.



RODOLPH CHARLES WINGFIELD.

[*Frontispiece.*